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SOUTHWESTERN MEDICINE

Volume XIV. JANUARY, 1930 No. 1

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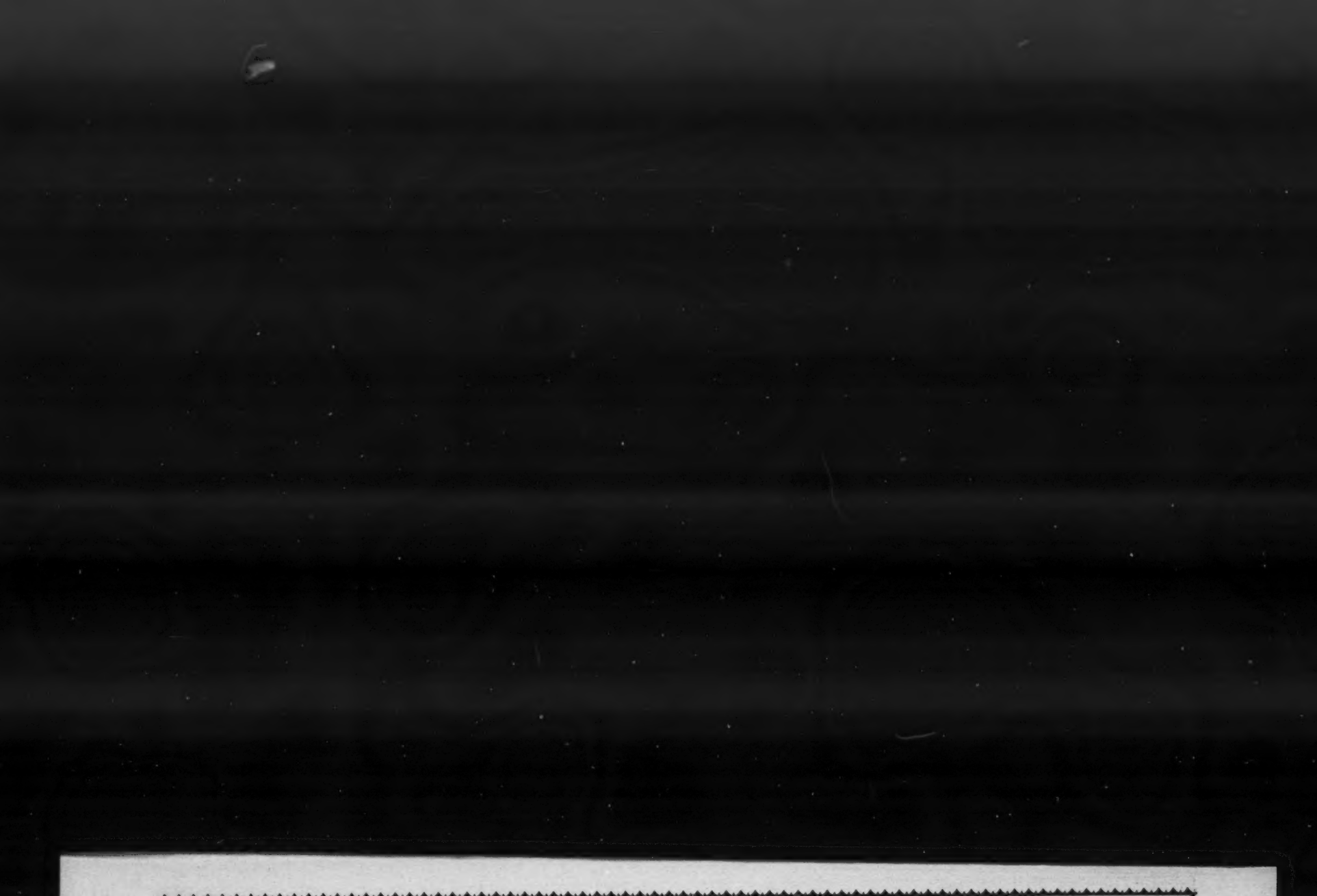

Southwestern Medicine

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PEPTIC ULCER

Limitations of Surgical and Medical Treatment

F. D. GARRETT, M. D.
El Paso, Texas.

(Read before the Medical and Surgical Association of the Southwest, at the fifteenth annual session, held at Phoenix, Ariz., Nov. 7 to 9, 1929).

Studying the more or less accepted indications for treatment of peptic ulcer, it seems to the writer that there is what may be called a "twilight zone," to which enough attention has not been paid. This paper is devoted to this field and to making the indications for medical and surgical treatment more definite.

It is agreed that medical treatment is indicated in simple uncomplicated peptic ulcer. Surgery is done: To make the diagnosis clear; to prevent cancer from developing on chronic ulcer; to repair perforations; to control hemorrhage; to relieve pyloric stenosis; to relieve the symptoms and prevent recurrence. I would like to consider each of these indications for surgery.

EXPLORATORY OPERATIONS

Most of us know that it is sometimes difficult to make a positive diagnosis, especially within the time usually devoted to examinations, even with the best of consultation and laboratory aid. If it is important for the internist to make a diagnosis before beginning treatment, it is even more important for the surgeon to do so, in my opinion, before undertaking an operation. In the case of the physician who has not made a positive diagnosis, there is always something that he can do to improve the patient's condition, meantime keeping the patient under observation. Within a period of ten days or two weeks a diagnosis can be made in many cases. The important point is that the physician shall have the knowledge, training and desire to study the patient and his symptoms very closely. In the case of the surgeon, what damage can be done, except in emergency cases, by waiting a few days or even a few weeks? But in many cases an operation is done without a diagnosis and the best interest of the patient has not been served. Blood is lost, there is a wound to heal, a scar remains, a certain amount of risk has been taken, and perhaps nothing definite accomplished. There are some surgeons, but not all, I am glad to say, who declare that one should not lose time with prolonged examinations. They would explore and see. But even with an open abdomen the pathological process, granted that the diseased organ is accessible, is frequently overlooked; and if found it may be impossible to make an exact diagnosis by inspection; for example, in gastro-intestinal hemorrhage. I will admit there are exceptional cases where exploratory operations are justifiable. But let us study our patients sufficiently long and carefully and at least have a clear conception of the problems involved and what the operation is to accomplish.

OPERATIONS FOR PREVENTION OF CANCER

Unquestionably a small percentage of cancers develop on old gastric ulcers. Cancer from duodenal ulcer is exceedingly rare. The combined reports from clinics where careful studies have been made indicate that cancer develops on about one gastric ulcer in twenty. In my observation and study of about four hundred peptic ulcers, of which some sixty have been gastric, there were only two cases of cancer which

developed after elective ulcer treatment and one of these had been urged to have an operation prior to treatment, but refused. We sometimes hear the assertion made that half of the stomach cancers have developed on old ulcers. This is not in accord with clinical experience. I think that the two diseases are confused by mistaking for an ulcer the necrotic area which develops on a carcinoma; in other words confusing cause and effect. All of us have seen patients who have suffered many years from ulcer of the stomach. They continued to live and did not develop cancer. Cancer of the stomach has a short history of digestive disturbance of rarely more than six months duration.

It does not seem justifiable to subject nineteen patients, who are in no immediate danger, to the risk of radical operation, in order to try to save one from possible cancer. I realize that if we are to save our patients from cancer it is necessary that operation be done early. In most cases careful examination and history will make the diagnosis clear. If cancer be found a carefully planned elective operation can be done. In passing I would stress three sources of information as being most important in the differential diagnosis between peptic ulcer and cancer; first, a good history; when a previously healthy person develops more or less constant gastric distress with rapid loss of weight cancer is probable. Second, the repeated finding of occult blood in the stomach contents and stools of a patient on a meat free diet is often a very early finding in cancer. Third, careful x-ray examination is of the utmost importance and many times gives invaluable information. I would refer you to Carman's classical description of the x-ray findings in malignant cancer. Reduced or absent hydrochloric acid and the presence of lactic acid are important but late symptoms.

OPERATIONS FOR PERFORATING ULCER

There is a vast difference between a perforating ulcer and a perforated ulcer. A perforated ulcer produces characteristic and unmistakable symptoms. The sudden onset of severe pain in the epigastrium, increasing rapidly in intensity, accompanied by epigastric tenderness, profound shock, and board like rigidity of the abdominal wall, in a person with ulcer history is almost diagnostic. There are two stages of perforating ulcer and the symptoms and indications for treatment are different in each stage. In the first stage the ulcer may have the muscle coat of the bowel for a base but the peritoneal coat is never ruptured. There is pouching at the base of the ulcer which gives rise to the x-ray niche so well shown in the films. The patient complains only of the usual ulcer symptoms and local signs of peritoneal irritation are absent. Operation is frequently advised for this type of patient on account of the supposed danger of complete perforation. In my experience this type of ulcer responds to medical treatment, careful diet, and rest in bed. The second type of perforating ulcer is accompanied by sharp intermittent, crampy pain, with localized rigidity over the site of the ulcer. There is complete absence of shock. A slight leak has occurred but not enough to cause more than localized reaction. The area of perforation may, and usually does, become attached to the omentum or adjacent viscera. Operation is indicated in this type of ulcer because of the possibility of serious complications. But it is well to remember that with rest, heat, oral starvation, and proctoclysis for forty-eight hours, some of these cases will recover under medical treatment if operation be refused.

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Some time ago opinion was divided as to the indications for surgery in acute hemorrhage from peptic ulcer. Today surgeons and internists agree that it is best to treat such cases medically because the operative mortality is very great. In my experience I remember only one death from acute hemorrhage of peptic ulcer. In cases of severe and repeated hemorrhages from an old fibrous ulcer it is best to wait for the interval between the attacks and get the patient in good condition before operating.

OPERATIONS FOR PYLORIC STENOSIS

In these days too much reliance is placed on x-ray findings and too little attention is paid to clinical symptoms. One half, three-fourths, or complete food retention at six or even twelve hours, means only that there is pyloric obstruction. It should be proven that the patient has permanent pyloric obstruction before he is subjected to operation. In our experience by far the larger percentage of cases of pyloric obstruction have turned out to be pylorospasm and could have been relieved by proper management and medical treatment. The subsequent treatment of the ulcer after pylorospasm has been relieved differs in no way from that of uncomplicated ulcer, except that rest in bed should be insisted upon in every case.

Cases of pyloric obstruction have very

marked hypersecretion. Before coming under the care of the physician their tissues are more or less dehydrated and the alkaline reserve in the blood has usually been increased. They are poor subjects for operation. By way of treatment, we give atropine and empty the stomach morning and night of the accumulated food and gastric juice. At night, advantage should be taken of this opportunity to introduce two or three ounces of warm olive oil before withdrawing the tube. If we are dealing with real stenosis the jejunal contents rapidly diminish and usually after five or six days become normal. It is possible to begin feeding small quantities of food the first day of treatment and alkaline control should be maintained during the whole twenty-four hours. These cases are peculiarly apt to develop alkalosis and it is important to give them large quantities of fluid in the form of normal saline with glucose per rectum or intravenously. If alkalosis with vomiting should occur the alkaline routine should be discontinued. If the obstruction does not yield to this type of treatment it should not be prolonged beyond a few days and the patient may now be operated with far better chances of recovery.

OPERATIONS FOR RECURRENT ULCER

Recurrence of peptic ulcer after surgical or medical treatment is due to the factors which produced the ulcer and favored its chronicity before the treatment. The treatment fails many times because the patient is led to consider himself cured too soon. If he is discharged at the end of three or six weeks after operation and a brief stay in the hospital with the idea that he is cured, and has no further oversight, the chances are that he will relapse. Treatment should take into consideration all of the contributing factors which may be present. Habits of living, social environment, personal habits and occupation, should be considered with as much care as focal infection, upon the clearing up of which we lay much stress. It would be unprofitable to discuss all of the theories of the cause of peptic ulcer, but there is one factor which is so common that it should be taken into consideration in all cases.

In about four hundred cases of peptic ulcer at least eighty-five percent have shown signs of nervous instability in the form of hyperacidity, hypermotility, pylorospasm, cardiospasm, or spastic colon. There were signs of unstable emotional tone and a tendency to overwork and overdo in all directions. They were often hard to control and keep on a prescribed routine. The success of any treatment, medical or surgical, depends upon the success of the physician in controlling the patient over a period of at least three years. Not that the case should end there, but during this time the changed habits should have become permanent enough to last during life. It is not enough to tell the patient not to smoke, not to drink coffee, liquors, or carbonated drinks and not to overeat. He must be shown why and be impressed that the chances of a relapse depend directly upon his faithfulness to the prescribed routine. He must be shown the importance of adequate rest and play, and learn to live the simple life free from worry. He should be taught to avoid tendencies to expend unnecessary amounts of nervous energy in any direction and how to relax at frequent intervals during the day. The nervous factor is responsible for the perpetuation of hyperacidity and when we have controlled this factor as nearly as possible the chances for relapse are minimum.

COMMENTS

It is not justifiable to do an exploratory operation to make a diagnosis unless a sufficiently long and careful study has failed.

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Pyloric stenosis is very much rarer than pyloric obstruction from spasm. Care should be taken to differentiate between the two conditions. Treatment over a period of several days makes the diagnosis clear.

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ILLUSTRATIVE CASES.

CASE 1. L. L., physician, age 52. Principal complaint, epigastric distress most marked one or two hours after meal.

Relevant past history; hard worker, smoker, coffee drinker, periodically alcoholic. Ulcer history dates back five years; symptoms more marked during last two or three months. Appetite is good but on a self restricted diet has lost ten pounds.

Principal points in physical examination indicate a poorly nourished, anemic patient; some fibrosis of lungs on account of old tuberculous process; epigastric tenderness. X-ray examination (Fig. 1) shows a large niche defect on posterior wall of stomach, with incisura opposite.

Diagnosis: Perforating ulcer on posterior wall of stomach at level of an incisura.

Treatment: Rest in bed. Modified Sippy. Patient's condition is satisfactory six months later, with some gain in weight. There is a strong probability of malignancy in this case.

CASE 2. G. R. H., farmer, age 26. Principal complaint of epigastric distress coming on about two hours after meals.

Past history of influenza in 1919; ulcer history dates back two years. Lately he has been vomiting at night and sometimes during the day; smokes; drinks coffee.

Physical examination shows a well nourished patient, very irritable and bad tempered. Breath foul due to extensive pyorrhea. X-ray examination (Fig. 2) shows niche on lesser curvature.

Diagnosis: Perforating ulcer on lesser curvature.

Treatment: Patient unmanageable. Would not enter hospital for medical treatment and refused operation. His wife carried out the treatment advised, she said, at home. He felt well for two years. He returned a few days ago saying that he had had trouble off and on for the past year. X-ray examination showed recurrence of ulcer. His mouth is still foul. No cooperation.

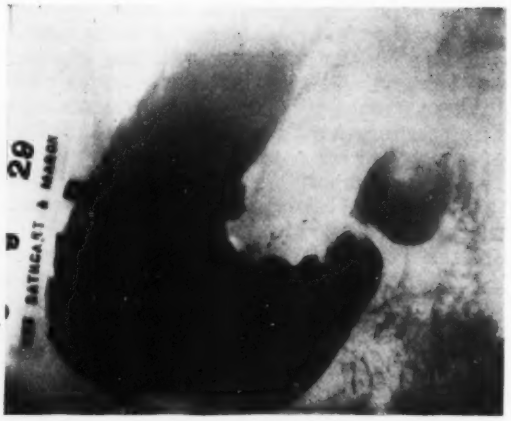
CASE 3. Sra. L. L., housewife, age 53. Principal complaint, epigastric distress with incomplete food relief; requires codein to relieve pain.

Past history of good health until six months ago, with more or less distress every day since.

Physical examination shows a well nourished patient, five feet in height. Marked epigastric tenderness and slight local muscla spasm. Free HC1 30 total acid 55. Occult blood in stools and stomach contents. X-ray examination (Fig. 3) shows niche on lesser curvature.

Diagnosis: Perforating ulcer high on lesser curvature.

Treatment: Operation advised but refused. Bed rest and modified Sippy carried out. Patient cooperated loyally and continues to do so. X-ray examination two and a half months after beginning treatment shows the ulcer healed. At the present time (six months later) she feels well.



THE SURGICAL ASPECTS OF GASTRIC ULCER

R. L. RAMEY, M.D.
El Paso, Texas

(Read before the Medical and Surgical Association of the Southwest, at its fifteenth annual meeting, held in Phoenix, Ariz., Nov. 7-9, 1929).

These cases should not be subjected to operation until they have had a fair try-out with a systematic medical regime. In cases which are not well marked we are sometimes puzzled to know whether or not we have a gastric or duodenal ulcer, cholecystitis or cholelithiasis, chronic appendicitis or whether the condition is due to extreme constipation or a simple hyperacidity. All of these conditions and others may have to be discussed. There are possibly few conditions in the abdomen which present so many different symptoms, and on the other hand, perforation of stomach ulcer is encountered in patients who have had little or no trouble at all. I recall several instances where the patients stated they had never suffered with their stomachs and could eat anything they wished. There are others, however, who have all the classical symptoms of ulcer but go on indefinitely.

It is not my purpose to go into symptoms, causes, etc., of ulcer but to give you in my short paper a brief report of my personal experience. When these cases show up for operation they have usually been treated and diagnosed by the clinician with the assistance of the x-ray man; except in acute cases consequently, will not discuss this phase of the subject. Surgery for ulcer of the stomach is as varied as the symptoms. Many men are doing extensive subtotal resections for ulcer of the stomach, combined with a gastrojejunostomy. Whether you do a simple resection or cauterize and invert the ulcer or whether you combine it with a gastrojejunostomy or a gastroduodenostomy, I think depends somewhat upon the conditions found after opening the abdomen. If I find a perforated ulcer or a non-perforated one for that matter, we excise it, repair the wound in the stomach and do no further surgery. This is especially true with a perforated ulcer, as you can not have a clean field with a ruptured viscus. A gastroenterostomy certainly would increase the mortality, so why add that additional danger and, too, I would consider it only necessary when the ulcer is situated at the pyloric opening or when there is a great amount of infiltration or scar tissue about the pylorus, causing an obstruction; also if you find multiple ulcers of the stomach. Formerly it was the custom to depend on a gastrojejunostomy curing the ulcer; at present it is rather the custom to excise the ulcer and do no further surgery unless absolutely necessary.

The most alarming conditions we have in ulcer are hemorrhage and perforation and to illustrate how uncertain the symptoms may be, I will report a case of each.

Not long ago a man was sent into the hospital suffering intense pain in his upper abdomen. We diagnosed his condition as perforation of stomach or duodenum; he refused operation for twelve hours, but finally consented. We found a perforation about the size of a pencil. The ulcer was excised, no further surgery done, patient making a good recovery gaining twenty pounds in a few months, showing he had evidently suffered a long time from his ulcer and did not know it. This patient claimed he had never had any trouble with his stomach, had been in the habit of eating any and everything.

Another case, a patient who had suffered from an acid stomach for several years but had not had any severe pain after eating or at any other time; had never had a hemorrhage. He consulted me and requested that I operate him for appendicitis as he was sure his trouble was due to this condition, since he had been told several times that he had a chronic appendicitis. I examined him and found no tenderness over the appendix nor any rigidity of the muscles and there apparently was no tenderness in his abdomen. Three days later this man died from a hemorrhage and autopsy showed he had two large ulcers on the lesser and posterior curvature of the stomach. I mention these cases to show how uncertain the symptoms are sometimes.

There are others whose symptoms are less well defined, who have persistent sour stomach, constipation, loss of weight. If these symptoms persist after proper medical advice, which I believe every man should have before he is subjected to operation, I feel an exploratory operation should be done and if no pathology is found with the gall bladder, appendix or other organs, even though you find no ulcer, you are warranted in doing a gastroenterostomy of your choice, and frequently get satisfactory results.

There are a few points I would like to emphasize from my own observation.

First, we should not delay an exploratory operation too long when there are persistent symptoms.

Second, all things being equal, if a gastroenterostomy is indicated I prefer one of the several methods of pyloroplasty as it is the more natural drainage for the stomach.

Third, a gastroenterostomy should not be

too small to give the stomach the proper drainage nor should it be too large as to empty the stomach too rapidly. Moynihan says that "the failure in a gastroenterostomy is usually because there was no organic lesion justifying it."

PERFORATING DUODENAL ULCER

J. W. HANNETT, M.D.,
Albuquerque, New Mexico

(Read before the Medical & Surgical Association of the Southwest at the fifteenth annual session, held at Phoenix, Ariz., Nov. 7 to 9, 1929).

The general recognition of duodenal ulcer is comparatively recent. According to Moynihan the first operation for chronic duodenal ulcer was performed in 1893, and the first successful operation for perforating duodenal ulcer in 1894. Hence, many practitioners now in their prime have not been impressed by the importance of possible ulceration in the so called chronic dyspeptic.

Bassler states that in four hundred thousand autopsies ulceration of the stomach was found in one per cent; and that in 59,940 autopsies of various series, healed or unhealed ulcer of the duodenum was found in 4.4 per cent. With these facts on stomach ulcer in mind we may expect duodenal ulcer four or five times more often than ulceration of the stomach.

No attempt will be made herein to deal with the various theories of the cause of duodenal ulcer. Our bickerings with the medical men as to the treatment and etiology of ulcer ceases to be a factor the very instant one of these ulcers perforate. In a review of the recent papers on chronic duodenal ulcer, one gleans rather meager information as to the danger signals of impending perforation. There is little comfort in blaming the medical men for a perforation after his so-called ninth cure. To my knowledge no one has as yet been able even remotely to state which of these chronic ulcers will remain cured after medical treatment, and which will perforate with or without medical treatment. One of my own cases perforated after several days in bed under a rigidly restricted diet. Another perforated a few weeks after receiving a clean bill of health from a clinic that we have perhaps all visited and held in high regard.

It is quite evident that we are in an era of indecision as to the proper and immediate treatment after a chronic ulceration has been diagnosed. Once a positive diagnosis has been made, right then a prompt decision as to treatment should be made. Our responsibility is immediately greater, and if for no other reason than for our own self protection, a full knowledge of his danger should be explained to the patient. He should be told that temporary relief, possible cure or ultimate perforation can be expected.

Dreyfus, in a recent work, feels that some duodenal ulcers are likely surgical from their very beginning. He frankly admits that we have no way of determining which of them will heal, and which of them when healed will leave a constriction that will demand surgical intervention.

What, then, should we advise these patients with chronic ulceration? If the surgeon could promise one hundred per cent cures with operation, the answer would be easy. And the same holds true with the medical man. In surgical interference, despite the excisions, short circuits and various plastic operations, in the most experienced hands, we still have our marginal ulcers, recurrences and even repeated perforations in rare instances.

The percentage of medical cures in actual figures seems to be rather vague. Some of the surgical clinics are claiming as high as ninety per cent cures in chronic ulceration. Perforation following operation is almost nil compared with perforation following treatment. Surgical mortality in the various operations for chronic ulcer in recent years is low. On the other hand, Deaver recently stated that twenty per cent of duodenal ulcers eventually preforate with a mortality of as high as seventy-five per cent. With the above preface and the calamity of perforation in the background, and with neither medicine nor surgery effecting a hundred per cent cure, would not the following compromise be fair to the patient, the medical man and the surgeon? A thorough medical treatment over a reasonable period of time with frequent clinical and laboratory examinations. Eradication of discoverable foci of infection and good general management. If symptoms and findings have not disappeared, recommend surgical treatment. Second, if all symptoms and findings do disappear and later reappear, again immediately interfere surgically.

With our limited knowledge of the physiology of the duodenum, it would seem good reasoning to conclude that in obstinate ulcers or recurrent ulcers, the cause has not been ascertained and that the alteration produced in the function of the stomach and duodenum by a plastic operation will effect the greater number of cures.

An apparent danger of medical treatment is the false sense of security that exists in the mind of the patient as soon as symptoms abate. With diet, rest, alkalies, he is

free from pain, and immediately forgets his diet and his rest, his medicine as well as his physician. Furthermore, duodenal ulcer, so frequent in the male, attacks him in the most active period of his life, and it is natural for him to revolt from diet or any restriction that tends to dominate his life. Were these facts not true few duodenal ulcers would need surgical treatment and perforations would be extremely rare. The patient may listen attentively to us while we are mapping out a six months to three years mode of living for him. Yet few will follow instructions after the abatement of symptoms.

In the diagnosis of chronic duodenal ulcer three features stand out in the majority of cases; history of pain in relation to food intake, high acidity, and duodenal cap deformity. Dreyfus states that the x-ray will show duodenal ulcer in ninety-six per cent of cases. However, it is well to bear in mind that the very first evidence of duodenal ulcer may be hemorrhage or perforation. Differential diagnosis of acute perforation without a positive history of preceding chronic ulceration is difficult to make. Yet there is perhaps no other acute abdominal condition wherein time is such an important factor. In this Western country we boast of our wide open spaces and magnificent distances, but the doctor in the town not equipped with hospital facilities, who is called to attend any acute abdominal condition, is confronted with a grave responsibility. To split hairs over a diagnosis will mean a life.

Briefly, the picture of an acute perforated duodenal ulcer is as follows; sudden acute pain in the upper abdomen and lower chest, rapid, short respirations, subnormal temperature, pulse not rapid in proportion to respirations, face grey and perspiring, the patient does not want to be examined, the knees are drawn up and the abdomen board-like in rigidity. In other words, the patient is sicker and appears sicker than in any other abdominal condition with the exception of acute pancreatitis, and acute pancreatitis is an extremely rare condition. In the past eight years the writer has operated ten cases of perforated duodenal ulcer and during that time has never seen a case of acute pancreatitis. The blood count in the beginning of the perforation is usually unchanged.

In speaking of the time element in perforating duodenal ulcer, W. J. Mayo informs us that a mediocre surgeon operating in the first six hours will have a much lower death rate than the master surgeon operating after eighteen hours. Up to the present time the patient with a perforating ulcer in an isolated community has been doomed. The advent of the aeroplane combined with more alert medical attention will save many of these cases in the future.

We all agree that immediate operation is necessary in acute perforation. The pendulum seems to swing back and forth concerning what should be done at operation. It would seem to be unwise to attempt to standardize surgical treatment for all perforating ulcers. The majority of surgeons at present seem to feel that a simple closure of the ulcer is the safest method. There are perhaps a small percentage of these cases, with the patient in good general condition, and a small amount of leakage into the peritoneal cavity, where a short circuit can safely be done. This is particularly true in the so called subacute cases. With the proper surroundings the gastroenterostomy has the double advantage of relieving the tension on the suture line of the closed ulcer as well as permanently curing the condition. In the great majority of cases simple closure of the ulcer is perhaps the safest. In my own cases cigarette drains have been installed. In the subacute cases, or in the cases of operation immediately after perforation, drainage is likely more harmful than helpful. Bacteriological examination of the stomach or duodenal contents taken at the operating table after recent perforation is usually sterile. This of course is not true in the late cases. A thorough aspiration of the abdominal cavity should of course be attempted. The use of the duodenal tube in the stomach after operation is particularly valuable in these cases.

The following series of ten cases of perforating duodenal ulcer have been operated by the writer in the past eight years. The high death rate has prompted me to write this paper. The fact that a recognized duodenal ulcer is allowed to go on to perforation is a reflection on us all. Medical men would do well to listen attentively to the history of patients with digestive disturbances and not pass them off too lightly. More stomach analyses should be made and more use made of the roentgenologist.

CASE 1. Farmer, age 34 years. Admitted to the hospital March 24, 1921. Diagnosis, acute appendicitis. Operated March 24, 1921, at which time appendix was removed and drain installed. The above case was operated and managed by another surgeon. April 18, 1921, twenty-five days after the above operation the patient was first seen by the writer, the attending physician being out of town. It seems this patient had gradually grown worse from the time of his operation twenty-five days previous. Examination showed the patient in extremis. A large mass filled the

whole of the upper abdomen to a point somewhat below the umbilicus. The patient had been vomiting for several days and was greatly emaciated. His bowels had moved with enemata. Diagnosis of localized peritonitis secondary to an appendectomy was made. The patient was given a blood transfusion and an incision made through the right rectus in the upper abdomen. A large amount of foul smelling pus and particles of food immediately came through the incision. Nature had made heroic efforts, over a period of several weeks, to wall off what must have been a small leaking perforation in the duodenum. While over one half of the abdomen was taken up with the abscess this patient had never developed a generalized peritonitis. The perforation in the duodenum was fully as large as a ten cent piece. The mucous membrane was pouting through the aperture. In conversation with one of the assistants at the previous operation, I was informed that the patient had rather an angry looking appendix and there was some milky fluid in the peritoneal cavity, but the appendix proper had not perforated. It is quite possible that this patient had a small perforation that was overlooked at the time of the appendectomy. An attempt at closing the perforation was made and drainage installed. The patient died within twenty-four hours.

CASE 2. Peace officer, age 31 years. Admitted to the hospital December 10, 1921. This patient was seen at his home at 4 p. m. on the day of his admission. He stated that he had had a severe pain in the abdomen that woke him up at two a. m., fourteen hours before being seen by me. It seems he had gone to bed an hour before, after consuming a large quantity of alcoholics and food at a wedding party. Various home remedies had been given him and by morning he was somewhat relieved. He had some abdominal distress during the day. He was sitting up in a chair fully dressed when first seen by me. Examination revealed a moderate amount of rigidity all over the right side and epigastrium. Pulse was 74, temperature 100°. This patient walked to an automobile and was taken about one mile to the hospital. His white blood count was 9,000. No red count or differential was made. Diagnosis of a moderately acute appendicitis was made. His abdomen was opened at 7 p. m., three hours after admission. An innocent-appearing appendix was removed. We were on the point of closing the abdomen when a small amount of milky fluid appeared in the upper angle of the incision. The incision was immediately extended upward and examination of the duodenum showed a plastic lymph exudate in the center of which was a perforation about the size of a pin head. There had been but little leakage of fluid into the peritoneal cavity. The perforation was closed, a posterior gastroenterostomy performed and cigarette drain installed. Patient was dismissed in seventeen days in good condition.

CASE 3. Section laborer, Mexican, age 49 years. Admitted to the hospital June 22, 1924. This happened to be in the middle of the afternoon on Sunday, and as the laboratory technician was not in, no blood count was made. The attending physician called me on the telephone, informing me that he had a case of acute appendicitis and that I should come to the hospital to operate. Upon my arrival the physician, absolutely sure of his diagnosis, had the patient in the operating room and partially anesthetized. Hence, no examination was made by me. Upon opening the abdomen a thin fluid was found free in the peritoneal cavity. The appendix appeared normal. Upon enlarging the incision we found a minute perforation in the duodenum with the usual surrounding exudate. The perforation was closed and a posterior gastroenterostomy performed. The patient was discharged in two weeks in good condition.

CASE 4. Retired meat cutter, age 59. Admitted to the hospital October 27, 1924 for fractured clavicle following a street fight after several days of alcoholic intoxication. After regaining his mental equilibrium he informed his physician that he had been suffering for a number of years with stomach trouble, and about the only thing that relieved him was drinking freely of alcoholics, further stating that as long as he was in the hospital he would like to have an x-ray examination of his digestive tract. On October 31, at eight in the morning he was given the usual barium sulphate meal. He was on the table at two p. m. for his six-hour observation when he was taken suddenly with a violent pain in the upper abdomen and lower chest. His physician and myself saw him at the hospital a few minutes after he was stricken. He was sitting in a chair in his room with rapid short respirations, perspiration pouring from his face which appeared ashy. His pulse was small, he refused to be moved, in fact, refused to be examined. From the high location of the pain a diagnosis of angina pectoris was made. Morphine and nitroglycerin were administered hypodermically. This patient was again seen by me ten hours later. At this time his symptoms were abdominal and we immediately suspected a perforation. He was taken to the operating room where the diagnosis was verified by the fact that the opaque meal was spread over the whole abdominal cavity and not confined to the intestinal tract. His abdomen was opened, and most of the barium meal immediately came through the incision. An attempt was made to aspirate the free fluid in the abdominal cavity. There was a good sized perforation in the anterior wall of the duodenum. A simple closure of the perforation was made and a tag of the omentum sutured over it. This patient died four days later of a general peritonitis.

CASE 5. Artist, age 25. Digestive disturbances for nine years. History of irregular treatment for syphilis. Admitted to the hospital at 2:30 p. m. on June 30, 1926. It seems that morning at 11:30 he was having his spine adjusted by a chiropractor for the cure of this digestive disturbance, when he was suddenly stricken with a violent pain in the abdomen while on the chiropractor's table. The chiropractor helped him to his automobile and drove him to his studio where copious amounts of sodium bicarbonate solution were given by mouth as well as several quarts by rectum. No relief. Then a medical man was summoned. When this patient was seen by me the abdomen was markedly distended and dull on percussion, with a board-like rigidity. His pulse was 80, mouth temperature 97 on admission to the hospital. His white count was 7,700, polys 56, small lymphocytes 40, faint trace of albumin in urine, and many granular and hyalin casts. Diagnosis of perforating duodenal ulcer was made in this case. Upon opening the abdomen upwards of a gallon of water and food particles came through the incision. There was no difficulty in locating the perforation as a stream of clear water spurted through the hole maintaining the level of two or three inches above the gut. The fluid was aspirated from the abdomen. We contented ourselves with a simple closure of the perforation. Small stab wounds were made in either flank and a cigarette drain from each side installed, as well as one in the upper incision. This patient was discharged from the hospital twenty-six days later. He now has an incisional hernia in the upper abdomen.

CASE 6. Clerk, age 20. Admitted to the hospital April 28 at 8 p. m. He had been having some digestive disturbances for several weeks. He was taken with acute cramp like pains in the abdomen about an hour before admission to the hospital. This patient had a typical board-like abdomen. His pulse was 88, temperature 98, white blood count 3,600, polys 58, urine negative. Perforating ulcer was suspected. Upon opening the abdomen of this patient we found a moderate amount of flocculent fluid with a perforation in the anterior wall of the duodenum about the size of the lead in a lead pencil. A simple closure of the ulcer was done. A stab wound was made in the midline above the pubes and a drain installed in the cul-de-sac, as well as a drain in the upper incision. This patient was discharged in thirty-one days.

CASE 7. Electrician, age 47, admitted to the hospital July 31, 1928 at 10 p. m. He had been seen by several doctors during that afternoon and evening. He had been taken with an acute abdominal pain about twelve hours before admission. He refused to go to the hospital, and had several hypodermic injections of morphine without much relief of pain. When called in consultation, we found this patient on the floor by the side of his bed with his knees flexed and in extreme agony. He had taken several enemas, and still refused to be taken to the hospital. This patient refused any attempt at examination of the abdomen, claiming that he could not bear to have his abdomen touched. About fourteen hours after his perforation his abdomen was opened and the perforation in the duodenum closed in the presence of a large amount of free fluid and partially digested food. One rubber tube and two cigarette drains were installed in the incision. This patient died of general peritonitis four days later.

CASE 8. Coal miner, age 22, admitted to the hospital December 8, 1928. This patient had been complaining of indigestion for the past year. He had a gastroenterological x-ray study one year before, at which time a diagnosis of duodenal ulcer was made. He was advised by his attending physician and myself that he should be operated. Operation was refused. His physician mapped out a diet and medical treatment, both of which he refused to follow. His attending physician saw him one and one half hours before his admission to the hospital. He was already sick with a mild influenza when he was taken with a severe pain in the epigastrium. This pain moved down the right side into the illac region, then across to the left iliac region. At the time of his admission his abdominal pain was general, particularly with respirations or any movement. He had absolute abdominal rigidity. Pulse on admission to hospital was 140, respiration 60, temperature 98. White blood count was 15,000, polys 90. Trace of albumin in urine. Two hours after this patient was stricken a right rectus incision was made through the upper abdomen. A little over one quart of fluid was aspirated. There was a perforation about the size of a lead pencil in the anterior wall of the duodenum. The edges were freshened and the ulcer closed and covered with a small reflection of the omentum. Stab wounds were made in the left and right inguinal regions, a drain installed in the pelvis and a cigarette drain between the stomach and liver. This patient died seven days later of pneumonia.

CASE 9. White woman, age 50, housewife, admitted to hospital December 9, 1928 at 10:15 p. m. Twenty hours before admission this patient was taken with an acute abdominal pain after eating a midnight lunch. A physician was not summoned until five or six hours later. Hypodermics of morphine were administered for pain. She was one hundred and thirty-five miles from a hospital and it was several hours before a train was due. Upon admission to the hospital her pulse was 110, respiration 38, temperature not recorded. White blood count 14,000, polys 89. Trace of albumin with hyalin and granular casts. Her pupils were markedly dilated and she was delirious. Her abdomen was considerably distended and tender. A tentative diagnosis of acute cholecystitis was made. Her abdomen was opened under local anesthetic. There was a small amount of cloudy fluid in the peritoneal cavity, appendix normal in appearance and a perforation found in the anterior wall of the duodenum. A simple closure of the perforation was made and cigarette drain installed. This patient died two days later. Her mental condition never cleared up. We were never satisfied as to the actual cause of her death, but felt that it was due to some cerebral condition.

CASE 10. This case was operated some two hundred miles from my home at a small hospital and the age and details are not available. This man was an automobile mechanic in his late forties. He was taken with an acute abdominal pain some sixteen or eighteen hours before operation. His physician had made a correct diagnosis of perforating ulcer before operation. Operation revealed a perforation in the anterior wall of the duodenum, with the usual amount of induration surrounding the ulcer. There was a considerable amount of flocculent fluid free in the abdominal cavity. The ulcer was closed and drainage established. The attending physician informed me that this man died three or four days later following a profuse hemorrhage from the stomach. No autopsy was done, hence no information available as to whether the hemorrhage came from the repaired ulcer site or whether there was another ulcer that was overlooked at the time of operation.

You will notice from the above reports that the ages of these patients vary from twenty years to fifty-nine, and that the perforations occurred at practically all seasons of the year. Cases two and three could likely be termed subacute perforations. Gastroenterostomies were performed in these two cases. Both of these cases had the shortest and most uneventful convalesences. Three of the acute perforations were operated within two hours of the attack; one of these died of pneumonia in seven days, the other two recovered. Three of the other cases which averaged about sixteen or eighteen hours from the time of the perforation to the time of operation, ended fatally. There is only one case in which a positive diagnosis of chronic duodenal ulcer had been made prior to the perforation. In this series there are nine males and one female.

CONCLUSION

Six deaths out of ten perforations is rather a gloomy record. Our only alibi is late surgical intervention after perforation. To escape from this mortality we must treat these cases successfully before they perforate.

DISCUSSION

(Papers of Drs. Garrett, Ramey and Hannett.)

DR. FRANK J. MILLOY, Phoenix, (opening):- It has been a pleasure to listen to the papers of Drs. Garrett, Hannett and Ramey. It is very evident that the medical man still favors medical treatment and the surgeon is inclined to surgical treatment.

Dr. Garrett has very conservatively defined the border line of ulcer cases whose treatment should be supplemented by surgery. Dr. Hannett has given us the clinical picture of perforated ulcer and has urged surgical treatment in order to eliminate this serious complication. He has mentioned Deaver's statistics which claim that twenty per cent of duodenal ulcers perforate sooner or later. The late Dr. Sippy's figures were one half of one per cent. Dr. Sippey may be low but it would seem that Dr. Deaver must be considerably high in his estimate, when we remember the fact that four per cent of autopsies show either healed or active duodenal ulcers, and we really see very few perforations. Dr. Hannett has seen ten cases and I can recall about three cases in my ten years of practice. If the average was one per cent, it hardly seems fair to subject 99 patients to surgery for the sake of one perforation. The average mortality for gastric surgery is considerably higher than one per cent. It must be remembered that we are speaking of the average operator and not the expert who performs operations on the stomach and duodenum several times daily.

Acute hemorrhage is never an indication for emergency operation. Rigid ulcer management will always arrest hemorrhage and it has been my experience that these are the medical cases which give the best medical results. The reason for this is that we have these patients in bed five or six weeks at the start and it is much easier to impress on such a patient the seriousness of continuing the treatment for a year or more.

There are several reasons for the failure of medical treatment of peptic ulcer. The commonest failure is probably mistaken diagnosis in taking histories, and inadequate examinations of gastroenterological cases. It is remarkable the number of patients, having digestive disturbance, who have at sometime or other been placed on some form of medical treatment for ulcer of the stomach. A great many of these cases have not been thoroughly examined and sufficiently studied, and do not have ulcer and have failed to get any relief from their symptoms, and the medical treatment for ulcer has been given the blame for failure. The failure to recognize associated appendix and gall bladder disease is another common occurrence. Fifteen per cent of patients with peptic ulcer have cholcystitis. These cases explain many of the so called medical ulcer cases which are classed as partially relieved and also many of the cases with return of symptoms. It has been my experience that a fair percentage of such cases will have a return of their ulcer distress even after a year's treatment. Another common failure of medical treatment is the fact that patients are not required to follow their medical treatment for a long enough period of time. A short period of a month or six weeks of medical treatment merely allows on ulcer to be filled in with granulation tissue. If an ulcer is to be permanently healed, this granulation tissue must be replaced by a normal mucous membrane which contains the normal amount of circulatory and nerve tissue. In order to accomplish this, the course of treatment should extend over a period of six months to a year and even longer in some cases.

If surgical treatment of ulcer could accomplish a high percentage of cures I am sure most everyone would welcome it. The reason that it does not accomplish better results is because it comes under the head of plastic surgery. The surgery may be either conservative or radical; it may vary from the simplest gastroenterostomy to a complete extirpation of the entire ulcer bearing area of the stomach and duodenum. It always seemed to me that the best way to decide such a question is to consider one's self in the position of the patient. Probably none of us would object to having a diseased appendix removed if we were the patient and I am sure we would feel the same way if we had gall stones, but I really believe the majority of us would consider medical treatment several times before we would have the lower end of our stomach removed or even as much as a gastroenterostomy.

One other point must not be overlooked in the supervision of patients with peptic ulcer and that is the general condition of the patient. Many patients are undernourished or are just sick either from lack of sufficient nourishment or from restricted diets. The human being is omnivorous in his habits. It is probably a serious error to place any patient, and this applies to all types of patient, including hypertension cases and nephritics, etc., on a restricted diet for an indefinite period. It is better after a certain period of specific treatment to forget the organic trouble and place a patient on a general diet containing meat and fruit and vegetables with plenty of vitamins. And after the general condition has improved and several pounds have been gained, return to specific diet again.

In conclusion, let me emphasize the importance of determining by careful study, the presence of associated abdominal pathology in recurrent peptic ulcer, and also the necessity of prolonged cooperation on the part of the patient whether the treatment has been medical or surgical.

DR. E. PAYNE PALMER, (Phoenix):-After listening to these splendid papers and discussion on duodenal and gastric ulcers, I feel that there is not much more to say than Amen, but I have inherited the dominant female trait of wanting to get in the last word.

Combined physical and mental exhaustion are causative factors in duodenal and gastric ulcers. These ulcers do not occur in drones. They are most frequently seen in young men who are hard workers and under mental strain. At the recent meeting of the American College of Surgeons in Chicago, Dr. George W. Crile gave a lecture on the "Influence of the Thyroid and of the Adrenals in the Production and Treatment of Peptic Ulcer." He believes that the over activity of the thyroid and adrenals as a result of this strain is responsible for these ulcers. He has cured obstinate ulcer cases by the removal of one adrenal gland and partial thyroidectomies. The catastrophes of duodenal and gastric ulcers are perforation, hemorrhage and malignancy. No one knows how many people have ulcers, therefore no one knows what per cent perforate or hemorrhage. Most of the statistics given are based on one individual's experience. If it is a gastro-enterologist, his percentage of the total cases seen is small. If it is a surgeon, his percentage of the total seen is large. The reason for this is that the gastro-enterologist sees a large number of cases that are under proper treatment and the surgeon sees many cases that have perforated and hemorrhaged. Most of them have not received proper treatment. In duodenal ulcers hemorrhage and perforation are more frequent, and it is believed that there is greater tendency toward malignancy in gastric ulcers but as yet there is no proof. All cases show more or less advanced chronic duode-

itis or gastritis in various stages. In five to ten per cent of cases ulcers are multiple, duodenal and gastric ulcers coexist.

The particular important question in ulcers, particularly gastric ulcers is, does carcinoma exist? Ulcers larger than 2.5 cm. in diameter usually show carcinoma. Subacute perforating ulcers are rarely carcinomatous. It is impossible to differentiate early carcinoma from simple ulcer by any known clinical means, including the x-ray. Operation should be undertaken when prolonged proper medical treatment has failed to obtain a cure. No one should use one type of operation for all cases; the factors to consider are location and character of the ulcer, the possibility of malignancy, the degree of acidity and the condition of the patient. Posterior gastrojejunostomy gives the largest percentage of cures, estimated at about 90 per cent. This procedure gives its most brilliant result in obstructing ulcers near the pylorus.

Balfour's cautery excision has many advantages. Finney's pyloroplasty is a splendid procedure in healed duodenal and gastric ulcers which obstruct the pylorus and duodenum. Deaver and others are removing the anterior half of the pyloric sphincter muscle with satisfactory results. Partial gastrectomy should be reserved for the saddle ulcers, the multiple ulcers and the ulcers with a suspicion of malignancy. The Billroth or Polya type of operation, or their modifications, are the operations of choice. The surgical treatment should always be made as safe and simple as it is possible with the least discomfort and risk to the patient. The operated cases should be returned to the medical advisor for constant care over a long period of time in order to secure satisfactory results.

DR. GARRETT, (closing):- I am glad to hear the medical and surgical sides of the ulcer question discussed in one symposium. I notice that neither claims to cure all. The surgeon operates and the ulcers return; the medical man treats them and they do not all get well. A certain percentage of them do not get well, whatever the treatment. I would like to emphasize one or two points. Dr. Milloy touched on the diet. It is certainly a mistake to restrict the ulcer patient too much. I do not think the diet is the biggest factor in the management of the ulcer patient; perhaps the habits are just as important. Many ulcer patients are smokers and they rarely get well, and the same can be said of other stimulants which increase the hyperacidity.

DR. RAMEY, (closing):- Just a point or two about the principle difference in the statistics of the surgeon and internist. The acute abdomen does not go the medical man but to the surgeon, and it is a different type of case that is handled by the two men. Dr. Hannett spoke of ulcers having to be differentiated from angina. It must be differentiated from many things, such as appendicitis, gallstones, pneumonia, angina,—all are simulated by perforating ulcer. As to the mortality; in the Michael Reese Hospital, I heard Lahey of Boston say that the mortality was seven or eight percent; I heard Finney, talking on pyloroplasty, say that the mortality was 4.8 percent. Dr. Palmer spoke of Crile; you get the impression from Dr. Crile, which of course he does not intend, that you might look for ulcer in every case of thyroid. He thinks that any condition causing hyperacidity will cause ulcer, and in exophthalmic goitre you nearly always have hyperacidity. If you have ulcer and hyperthyroidism, do the thyroidectomy and in many instances the ulcer will get well. The type of operation will depend on what you find when you get into the abdomen, using your judgment after you see the pathology. After gastro-jejunostomy, patients not uncommonly have ulcer atth e stoma with stasis in the loop of bowel.

DR. HANNETT, (closing):- With regard to gastro-enterostomy in perforation; in many cases there is induration around the ulcer and in such cases I have not cared to do pyloroplasty. With regard to nature's effort at closing the perforation in ulcer; have never seen any attempt at closure of the ulcer from the outside in any one of these ten cases. I would agree that all ulcer cases should have medical treatment and know that many of them are cured. The unmanageable cases which refuse medical treatment, or the suddenly perforating cases, are the ones which frighten the surgeon and cause us to lean a little toward surgical treatment.

SOME OF THE LESSONS TAUGHT BY NECROPSY IN PULMONARY TUBERCULOSIS

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(Read before the twentieth annual meeting of the Pecos Valley Medical Association, held at Roswell, N. M., Oct. 10, 1929).

Importance must be attached to the relationship that exists between necropsy findings and antemortem diagnosis and treatment in all diseases, and pulmonary tuberculosis is no exception. It is the surest way of learning our mistakes and of obtaining means of avoiding them in the future. At the United States Marine Hospital, Fort Stanton, New Mexico, where only tuberculous patients are treated, a post mortem examination is made on about ninety per cent of the deaths. This is an advantage which few sanatoria have. The medical staff at Fort Stanton realizes this and tries to profit by it as much as possible.

In this paper some of the lessons which have been learned therefrom will be given. These are drawn especially from the last seventy autopsies. Only pathology that has some direct bearing upon the points to be brought out, will be discussed. All unnecessary details and irrelevant facts will be purposely omitted. The findings are based upon the gross pathology for the most part, but a microscopic examination was resorted to whenever it seemed necessary to throw more light upon the subject. All of the patients are beneficiaries of the United States Public Health Service and are, therefore, adult males, and furthermore, all of them that died had pulmonary tuberculosis. The lessons to be emphasized will be taken up separately under the various organs, beginning with those of the chest.

PLEURA

The pleura, which is involved in a large number of cases of pulmonary tuberculosis, is of great importance in this disease. Upon its freedom from extensive adhesions de-

pends the result of our pneumothorax therapy. In this study it was found that in only eight instances was the pleura free from adhesions (less than six per cent). The underlying pulmonary pathology was checked up in these cases and found to show a small cavity in one, some tuberculous infiltration and caseation in two, and scattered healed tubercles in four. Then there were twenty-eight other pleurae in which only slight or moderate adhesions, apical or diaphragmatic for the most part, were present. This gave a total of thirty-six cases in one hundred and forty (about twenty-six per cent), which it was thought might have allowed a suitable pneumothorax collapse of the lung. Perhaps none, or very few, of the remainder would have admitted satisfactory pneumothorax treatment as they showed more or less extensive adhesions, varying from dense ones over the upper lobe only, to a complete obliteration of the whole pleural cavity.

Comparisons of the pleural condition of the cavernous lungs and those without excavation, were interesting. There were only twenty-seven non-cavity lungs, and eighteen of these (or sixty-six per cent) were represented by the thirty-six least diseased pleurae given above as suitable for pneumothorax. In the cavity cases, the strongest adhesions and the thickest pleurae were found and when these were localized they occurred mostly in the vicinity of the excavations. This shows that in these patients at the time of death the condition would have prevented a satisfactory pneumothorax treatment in the great majority. The lesson taught here is that, when an induced pneumothorax is indicated, a prolonged period of observation is not feasible. This applies particularly to patients who have cavities. Our statistics show that dense pleural adhesions will develop in such cases sooner or later. This lesson is of the greatest importance to our patients and should not be overlooked.

LUNGS

Cavities. Passing on to the lungs, the most striking feature shown was that the x-ray film of the chest showed the pathology found at the postmortem faithfully. This was especially true of intrapulmonary cavities. Having the last film on exhibition at the postmortem, there could be no mistakes in these findings. In every instance in which an annular shadow was seen in the film, it could be accounted for by an intrapulmonary cavity of like location in the lung. This was found to be so, even of those annular shadows without any marked central rarefaction. In no case could any of them be attributed to either a localized pneumothorax or pleurisy (so-called pleural rings), of which we have read so much, as a possible explanation for some roentgenographic annular shadows.

The postmortem records show forty-eight bilateral cavity cases, seventeen unilateral cavity cases and five without cavities. While not all these were demonstrated in the x-ray, and those shown were not always as extensive as found at the autopsy, still ninety-four per cent of them were diagnosed roentgenologically. With the exception of one where a thickened pleura obscured a cavity shadow, it is possible that most of those that were missed had developed after the last x-ray film of the chest was taken, as these patients were too sick during the last months of life to permit a roentgenogram to be made.

On the other hand, when the physical findings were compared with the necropsy data, it was found that only fifty-eight per cent of the cavities had been correctly diagnosed in these terminal cases. Furthermore, there had been erroneous signs of cavitations in eight per cent. These could be accounted for at the postmortem by fibrosis or consolidation. There were no erroneous findings by the x-ray except in one case, where a large emphysematous bleb at the base showed as a thin-walled annular shadow. This was an atypical pear-shaped shadow and its cause, in the future, should be recognized.

So we have learned that x-ray annular shadows should be interpreted as representing pulmonary cavities. We can never be certain that a patient has not a pulmonary cavity until the physical findings have been checked up by the roentgenogram. Then, when an annular shadow or other signs of cavitation are seen, it must be considered that the patient has a definite cavity, no matter what the physical signs indicate. This is one of the most valuable lessons that we have learned at the postmortem table.

Hemoptyses. There were fourteen deaths from pulmonary hemorrhage. Nine of these died during the hemoptysis, and five as a result of a broncho or lobar pneumonia shortly afterwards, no doubt set up by infection from the blood in the respiratory passages. No attempt was made to find out whether these pneumonias were of tuberculous origin or caused by secondary infection. In the text books on pulmonary tuberculosis the mortality from hemoptysis is put down at a very low figure. Fishberg

gives it as two per cent. Much must depend however upon the type of disease treated. At Fort Stanton, including even small hemorrhages, our mortality is well over eight per cent. Also, the frequency of hemoptysis with us seems high. This we do not attribute to the altitude, as Dr. F. C. Smith, a former Commanding Officer, showed that barometric pressure and humidity were not causative factors. It is, no doubt, the type of the disease that alters the mortality. A large number of our patients are far advanced consumptives, as are probably many who come to the Southwest for their health. All the men dying of hemoptysis at this hospital had extensive bilateral or unilateral cavitations. The bleeding seemed to originate in these cavities, in all in whom this point could be ascertained at all.

A note on the status of artificial pneumothorax in the treatment of hemoptysis seems indicated here. We have used it only when the routine treatment had definitely failed. It was tried in five such cases. While it was a life-saver in two of them, who are still doing well at present, the other three were failures. One of them died of an uncontrolled hemorrhage in spite of the compression and the examination after death showed that the bleeding came from an uncollapsed cavity of the upper lobe. The other two contracted fatal pneumonia in the contralateral lung after the hemorrhage had been checked by the pneumothorax.

During the past three years, in which these autopsies were done, there were three deaths from pneumonia following about one hundred large or moderate hemoptyses in which artificial pneumothorax was not tried, and the above two among the five in which the pneumothorax was used to stop the hemorrhage. While the number is small, still a warning note seems warranted as to the danger of a therapeutic pneumothorax setting up a pneumonia in these cases. This is, no doubt, due to the forcing of blood and infected secretions from the collapsing into the uncompressed lung. This happens especially because much higher pressures must generally be given to control the bleeding than are used in the ordinary case. The third lesson, therefore, is that pneumothorax therapy should be reserved as a heroic measure in pulmonary hemorrhages, to be used only when other means have failed and it appears that the patient will die if left alone. To use it earlier carries the risk of having the patient die unnecessarily of pneumonia.

HEART

The most interesting point here was the small size of this organ in so many of the patients. A small, flabby, atrophic heart is the rule in far-advanced phthisis and this is generally in proportion to the great emaciation and muscular atrophy of the rest of the body. Thus, a small drop type of heart in the x-ray film should generally mean a poor prognosis.

INTESTINES

There were twenty-four patients with intestinal tuberculosis. This was the most frequent tuberculous complication found. The lesions were localized in the jejunum, ileum and colon. The part most frequently diseased was the terminal ileum. In eight cases, both large and small intestines were involved; in eleven, the small intestine alone. One patient who had been under treatment for intestinal tuberculosis for about eighteen months, showed several scarred areas as of healed ulcers. He also had a few small active ulcers. In regard to the frequency of this condition and the site of greatest involvement, it must be said that the intestines were not opened in every case, and it is possible that some ulcers may have been overlooked. This applies mostly to the large bowel, as ulcers in the small gut generally extend to the serosa and are easily seen and felt from the outside. However, the important point was that an antemortem diagnosis was made in only eleven of the twenty-four patients, forty-six per cent. Most of the others did not complain of any definite symptoms. In some of these, rather extensive ulcerations were present but, for the most part, those with the greatest pathology gave manifest clinical symptoms. We should endeavor to make a diagnosis of this serious condition whenever it exists. If this end can be attained early enough, the treatment should be beneficial to the patient. As Brown and Sampson have shown that intestinal tuberculosis will give characteristic x-ray findings with the barium meal, it is important to do this examination on all in whom there is the least suspicion of an involvement of the intestine. Earlier diagnosis, in which the use of the x-rays will help us, is the lesson which these deaths teach.

APPENDIX

This is an organ which is prone to give trouble in the tuberculous, as it seems very susceptible to acute inflammation which is quickly followed by rupture and peritonitis. Four of our deaths were due to appendicitis. In two, general peritonitis followed rupture. One of these was operated less than eighteen hours following the onset, but it was too late. In the other, a general peri-

tonitis developed while waiting for the acute inflammation to subside before operating. The other two patients died of intestinal obstruction caused by adhesions complicating an acute appendicitis. In one, the appendix which had ruptured, had been successfully removed more than a week. In the other, the adhesions developed around a localized appendiceal abscess. During the above period, fourteen other patients were operated upon for appendicitis. In one, there was a general peritonitis or rupture about twelve hours after onset. In four others, the appendix was gangrenous or ruptured.

In the seventy postmortems, we found three appendices which were badly inflamed and contained pus. Two others had definite gross tuberculous lesions. Four showed extensive adhesions as evidence of severe previous inflammation. In one of these, the appendix was almost completely buried in the terminal ileum. This patient's history showed a very severe attack of appendicitis which was suddenly relieved just before he passed a large amount of purulent material by rectum. The appendix had evidently ruptured into the bowel.

There were many other cases with adhesions in the appendiceal region showing the frequency of acute or chronic appendicitis. From our clinical experience an acute fulminating appendicitis is not a rare condition in phthisis. These patients should be operated on without delay as rupture seems likely to occur. While we learn to do as little surgery as possible in patients with pulmonary tuberculosis, we make an exception of the appendix and operate as soon as there are definite signs of disease.

KIDNEYS

There were twelve patients with gross tuberculous lesions of the kidneys at the necropsies: eight bilateral and four unilateral. Of the bilateral, six were extensive and two of moderate degree; while of the unilateral cases, one was extensive and three moderate or minimal. In nine of the twelve, the bladder showed tuberculous ulcers, and in two, the ureters were also diseased. The examination of the bladder was overlooked in one instance. As all of the other bladders examined were non-tuberculous, it is unlikely that many cases of renal tuberculosis were missed.

In two patients with tuberculosis of the kidney, this complication could be attributed as the direct cause of death, as their pulmonary disease was such as to be compatible with longer life. In one, little active pulmonary pathology was found besides a moderate size cavity in one lung. The other was an old fibroid type. In both of these, the localization of the renal lesion had not been determined. This was due to the fact that a cystoscopic examination had not been made. The fibroid type, who suffered severely before death, had extensive unilateral renal tuberculosis as far as a close gross examination revealed. This man could have gotten marked relief and his life been prolonged by an early nephrectomy. The other case was a bilateral one, but the pathology was much greater and of undoubtedly longer duration on one side. If it is true, as many authorities hold, that renal tuberculosis is primarily a unilateral disease, this man too, might have been diagnosed in time and a nephrectomy might have helped him.

These two autopsies have taught the lesson that the urinary organs of all tuberculous patients should be carefully examined and a cystoscopy done whenever indicated. We are already noting the benefit from these examinations. Another reason why we are checking up more thoroughly on the renal conditions of our patients is that only eight of the eleven renal tuberculosis cases had been diagnosed before death.

LARYNX

There were twenty cases of tuberculosis of the larynx, which made this the second most important tuberculous complication. All had been diagnosed before death, but many of them were diagnosed only after it had become a serious complication. In at least seven, it seemed be the most important contribution to the fatal outcome, because the pain exhausted the patient by preventing the taking of sufficient nourishment and by the loss of sleep. Attempts should be made to recognize the laryngeal involvement of our patients before it has become a menace to life. This can be done by a routine examination of the larynx of all phthisical individuals at regular intervals of, say, every two months. The early diagnosis, which is possible only in this way, followed by treatment with the electric cautery or heliotherapy and silence, should be rewarded by an arrest of the disease in many patients. Left unrecognized until far advanced, it soon becomes of greater danger to the patient than his pulmonary condition.

MENINGES

One of the other frequent complications was tuberculous meningitis, which, always fatal, accounted for seven of our deaths. The diagnosis was always easily made and the duration was from two to four weeks. One of these cases followed an epididimo-orchectomy, which seemed to have been a factor in precipitating the meningitis,

which occurred twelve days after the operation and was of twenty days duration. Meningitis should, therefore, be considered as a possible outcome in the operative treatment of tuberculous epididymitis and epididymo-orchitis.

SUMMARY

The autopsies of seventy patients dying of pulmonary tuberculosis showed that they could be divided into two classes: twenty-seven with tuberculosis of the lungs alone; forty-three with pulmonary tuberculosis complicated by tuberculosis of other organs. It was found remarkable that, of the twenty-seven with uncomplicated pulmonary tuberculosis, as many as twenty-one had some contributory cause to account for the death: viz, six, hemoptysis; six, pneumonia; four, spontaneous pneumothorax; three, cardio-renal disease; and two cases of fatal appendicitis. Of forty with extrapulmonary tuberculous lesions, the following distribution is tabulated:

Tuberculosis of intestines	24	34%
larynx	20	29%
urinary organs	12	17%
peritoneum	8	11%
meninges	7	10%
genital organs	7	10%
bones and joints	5	7%
liver	2	3%
spleen	1	1.5%
adrenals	1	1.5%
pericardium	1	1.5%

Thus, it is shown that one of the important features of this study was the great frequency of a tuberculous complication, or of an intercurrent disease, as the primary factor in the mortality. This was true in over two-thirds of the deaths, or sixty-seven per cent. Most of these have been reviewed. They teach us the importance of looking beyond the pulmonary field in all of our patients; and one of the problems we have before us in the reduction of our mortality is the proper diagnosis and treatment of complications which are extrapulmonary. We should not rest satisfied with an expert diagnosis of the pulmonary condition to the exclusion of important pathology in other organs. A special search for tuberculosis or other diseases of these other systems of the body should be carefully undertaken. In this class of patients, the alimentary, the genito-urinary, and skeletal systems are of particular significance. Individualization of our phthisical patients, with a constant lookout for possible complications, should be the rule.

CONCLUSIONS

1. It is realized that the number of these autopsies is too small to draw definite conclusions, yet they seem to point out some lessons which are well worth remembering. Just as the early diagnosis of pulmonary tuberculosis is paramount for its successful treatment, so are the early diagnosis and treatment of its tuberculous complications.

2. In laryngeal tuberculosis, an early diagnosis, before it has reached the stage where it is a menace to life, is essential. Do we always make our diagnosis early or do we wait for severe complaint from the patient before examining the larynx? I believe that more frequent examinations of the larynx of every patient should be practiced. This is particularly true among our out-patients, who have less medical supervision. At any rate, this complication should be recognized early enough for heliotherapy or cauterization to be of value. Otherwise, the prognosis must be extremely bad.

3. In renal tuberculosis, a greater effort at early diagnosis should be made. Cystoscopy and the laboratory examination of the urine at the very earliest suspicion of urinary disturbances, should be the rule. If we are not competent to make a diagnosis, including the localization of the renal tuberculosis, a specialist in urology should be called in consultation to examine our suspicious cases. When a unilateral diagnosis is made, a nephrectomy should follow if the patient's condition permits.

4. In intestinal tuberculosis we are confronted with the same problem of early diagnosis. Under proper treatment with ultraviolet light, proper diet, and calcium chloride intravenously, we have seen some of our cases improve greatly. One case, under treatment over eighteen months, showed signs of healed ulcers at the post-mortem. This can occur only if treatment is begun before too much destruction has taken place. At the slightest complaint of gastro-intestinal disorder, an examination with the barium meal should be made. Following the technic outlined by Brown and Sampson of Saranac Lake, the roentgenologic examination may reveal the diagnosis before this is possible clinically. In our necropsy reports, we have seen that fifty-four per cent of the cases of tuberculosis of the intestine had not been recognized during life, indicating that many of them did not show any characetistic symptomatology. This is the reason the x-ray examination is so strongly advised.

5. We should always be on the lookout for symptoms of acute appendicitis in phthisis, in order tooperate before rupture has occurred. During the last three years, at Fort Stanton, there have been an unusual number of acute fulminating cases of appendicitis among our patients. These pa-

tients should not be watched too long before operating, as we have learned by sad experience. While the policy of as little surgery as possible in pulmonary tuberculosis is a sound one, it should not be made to apply to the appendix.

6. Artificial pneumothorax in the treatment of hemoptysis is to be reserved as a last resort where the routine treatment has failed. In its use there is always the risk of pneumonia in the contralateral lung. It should, therefore, never be looked upon as a primary measure for controlling pulmonary hemorrhage.

7. Pneumothorax therapy, on the other hand, should be started as early as possible when indicated in suitable cases. To wait is to encourage the formation of pleural adhesions which may interfere with a successful result. This applies particularly to patients with cavities, as is shown in the pleural statistics fo these necropsies. Adhesions were more frequent and more extensive in the cavernous lungs.

8. Annular shadows seen in the x-ray films of the chest were due to intrapulmonary cavities in all of our postmortems. None were found to originate from a pleural condition, either a localized pneumothorax or a pleural ring. The x-ray showed at least ninety-four per cent of the cavities present at the time of death, whereas, only fifty-eight per cent could be found by the physical examination. No case of pulmonary tuberculosis should be treated without an x-ray film of the chest.

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SOME EXTRATHORACIC TUBERCULOUS LESIONS IN CHILDHOOD*

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THE ABDOMEN

Tuberculous mesenteric lymph nodes have been detected at the postmortem table or during exploratory operations, and seen in all stages of the disease from the recent progressive lesion to the highly calcified process. Opie's postmortem work has been an outstanding contribution to this subject. An advance in diagnosis was made by Dunham and Smythe in 1926 when they examined 120 school children, most of whom were under six years of age, many of whom gave definite history of exposure to tuberculosis, and all of whom reacted positively to tuberculin. They showed that calcified mesenteric lymph nodes could often be detected by x-ray examination. Indeed, seventeen per cent of these children manifested evidence of such conditions. Recently Soper and Dunham reported the results of their studies on 301 children from six to thirteen years old, most of whom reacted positively to tuberculin. Among the 301 children, evidence of calcified mesenteric nodes was found in sixty-four. Of the sixty-four, all but one reacted positively to tuberculin, and in twenty-nine of them definite exposure was known to have existed. In the detection of calcified lymph nodes, they made antero-posterior films in every case and lateral films in most cases. The lateral films they find "serve to confirm the anteroposterior films, to exclude the possibility of the calcium deposit being in the urinary tract through its being visible anterior to the vertebral column. An occasional lateral film reveals the calcification where it is overlooked or invisible in the antero-posterior film." They use thirty milliampere tube; fifty milliamperes; a distance of twenty-six inches; a spark gap of four inches for the anteroposterior and five inches for the lateral films. The exposure for the antero-posterior films is one-quarter of a second for a child seven years, and one-third of a second for a child of twelve years. For the lateral films the exposure is from one-quarter to three-quarters of a second. They employ an intensifying screen. Associated with the calcified mesenteric nodes they found tracheo-bronchial or lung disease, or both, in forty of the sixty-four cases. Cervical adenitis was present in three; tuberculous peritonitis in one and phlyctenular conjunctivitis in two. Recently one of the children in the group studied by Dunham and Smythe, who had evidence of calcification in the mesenteric nodes for several years, was accidentally killed. At the postmortem examination, the x-ray finding was confirmed and in addition, caseation was present.

These studies open a new field in routine examinations. The finding of evidence of calcification of the mesenteric lymph nodes may explain positive tuberculin reactions in patients in whom no other lesion can be detected. They also are an indication for treatment, as caseous nodes may rupture

into the peritoneal cavity, resulting in more extensive disease. Perhaps they open a new field in the prevention of tuberculosis, being potential dangers, as tuberculous tracheobronchial and hilum nodes are such from the standpoint of spreading bacilli to others.

Tuberculosis of the intestine is not common in childhood. When present it is usually accompanied by caseous lesions of the tracheobronchial and mesenteric lymph nodes. The intestinal lesions usually are secondary; however, primary lesions may appear among those taking huge quantities of tubercle bacilli into the digestive tract.

From infancy through the period of childhood **tuberculous peritonitis** is common during the third year of life. When the disease exists, the liver and spleen are frequently involved. In one form (ascitic) slightly cloudy or transparent fluid fills the peritoneal cavity. If intestinal adhesions develop, they are not dense enough to hold the parts together. In such cases miliary tubercles are found over the whole peritoneum. In the other form (tabes mesenterica) only a small amount of purulent fluid is found. The adhesions are so dense as to bind firmly the loops of intestine. Tuberculous ulcerations are usually found in the intestine.

The diagnosis of tuberculous peritonitis is sometimes aided by simultaneous occurrence of pleurisy. The symptoms often are of little avail in diagnosis since they may simulate those of so many other diseases, such as intestinal obstruction, strangulated hernia, appendicitis and typhoid fever. A markedly positive tuberculin test is of considerable value. One should always search for evidence of tuberculous lesions elsewhere in the body, such as those of the childhood type in the lungs, hilum and tracheobronchial lymph nodes and in the mesenteric nodes. A history of prolonged exposure to a tuberculous patient or the use of contaminated foods is important.

In the progressive stage of the disease, one should insist upon strict bed rest with comfortable atmospheric conditions. If intestinal lesions are absent, a general diet suffices. If they are present, the diet should be free from rough and irritating substances. In any event, cod liver oil and tomato juice should be administered after meals. The dose is to be consistent with the age of the child. That for an adult is one-half ounce of cod liver oil and three ounces of tomato juice. Heliotherapy by natural sunlight or artificial lamps is beneficial in many cases. The Rollier method for natural sunlight suffices. If alpine lamps are used, general exposures of one minute with the lamp at a distance of thirty inches from the body once or twice per day and increasing one minute per day if no reaction develops, are very valuable. In some cases it is desirable to expose only the abdomen; in others, on general irradiation, additional exposures over the abdomen are well tolerated.

The prognosis in many cases of tuberculous peritonitis is good provided the treatment is continued sufficiently long.

Prevention consists of avoiding further exposure to tubercle bacilli and the proper treatment of all children with tuberculosis of the childhood type, the pleura and mesenteric lymph nodes.

Tuberculosis of the kidney usually is secondary to disease elsewhere in the body. Among definitely tuberculous children the incidence of renal involvement is fairly high. Some have placed it at twenty-three per cent. Its diagnosis is not always easy but a definitely positive cutaneous tuberculin test is of considerable importance, as well as the finding of a tuberculous process in some other part of the body. The presence of tubercle bacilli in the urine confirmed by animal inoculation, is our best evidence. Catheterization of the ureters aids in determining whether the bacilli are being discharged from one or both kidneys.

The treatment of unilateral renal tuberculosis is surgical. But this should be followed by a long course of dietetic and hygienic treatment, with the addition of heliotherapy. Without surgery, tuberculin therapy is advanced by some workers.

The immediate prognosis is good with surgery, and postmortem workers have reported that spontaneous healing by fibrosis and calcification is not uncommon.

THE EYE

Conjunctivitis is one of the common forms of tuberculosis of the eye. This may be primary or secondary. Other parts, such as the retina, choroid, sclera, the iris and even the lachrymal glands, have been found to be tuberculous. The diagnosis of all forms of ocular tuberculosis is not particularly easy. In those parts easily accessible, such as the conjunctiva and lachrymal glands, direct smears are helpful. In each form of tuberculosis of the eye, one should make careful stereoscopic films, and place particular emphasis upon the nodes receiving lymph from the eye. Many cases of ocular tuberculosis are secondary and hence the focus should be sought. It may be in the tonsils, in the lungs, or elsewhere in the body. In addition to the general examination, one should always apply the intracutaneous tuberculin test and if this is negative when the dosage

has been increased to one milligram, the suspected condition most likely is nontuberculous. If the tuberculin test is positive and the patient manifests a rather strong hypersensitiveness, one should watch the focal reaction about the suspected lesions. The treatment of tuberculosis of the eye consists of the general measures employed in treating tuberculosis in any part of the body and general heliotherapy. By the use of tuberculin; excellent results may be obtained provided there is not an extensive and progressive tuberculosis in the lungs. The initial dose should never be larger than 0.001 mg. The increase should be very gradual, at four of five days intervals, until one milligram is used as the maximum dose. In severe cases, where there is a marked hypersensisitiveness to tuberculin, the initial dose should be even smaller; 0.0001 mg. will suffice and the increase of dosage should be very gradual. Most excellent results may be obtained in ocular tuberculosis by this method of treatment.

A close relationship exists between **phlyctenular conjunctivitis** and tuberculosis. In phlyctenular kerato-conjunctivitis, the etiological factor is practically always a tuberculous focus somewhere in the body and the conjunctivitis is due to hypersensitiveness to tuberculosis. As long as the high degree of hypersensitiveness continues, the conjunctivitis presists. The repeated attacks may last for months and even years. If the hypersensitiveness to tuberculin wanes, the attacks subside, but if there is a spread of the disease about the tuberculous focus already present in the body or there is a reinfection from the outside, the hypersensitiveness increases and the conjunctivitis returns.

The treatment consists of desensitizing the patient to tuberculin by subcutaneous tuberculin therapy. One should be extremely careful not to use enough tuberculin to produce a reaction either local or constitutional; O. 1 c.c. of a solution of 1:1,000,000 of old tuberculin is the initial dose. The dosage is increased very gradually every third day until 5.0 mg. (o.5c.c. of 1:100 dilution) of old tuberculin is used. Remarkable results are obtained by this method of treatment.

THE SKIN

Erythema nodosum is rather frequently associated with tuberculosis. It usually appears while the hypersensitiveness to tuberculin is marked. Dr. Stefano reported twenty-three cases of erythema nodosum in children from three to twelve years. In every case the tuberculin skin test was positive and markedly so in fifteen. To be sure, erythema nodosum is not always caused by tuberculosis; there are numerous other causes. In fact, erythema nodosum may be seen before clinical tuberculosis has been diagnosed. This is not an uncommon occurrence during the period of childhood. Again, it may occur while a clinical case of tuberculosis is under observation and treatment. When this is true, it often indicates an extension of the disease. Therefore, when erythema nodosum appears, whether the patient is one who has always been well or one who has previously suffered from tuberculosis, careful observations should be made for a new lesion or the extension of an old one. It may be followed by enlargement of the lymph nodes, peritonitis, pleurisy with effusion, meningitis, pericarditis and arthritis due to tuberculosis. In other cases there are no sequelae. Since these conditions often occur in children, it is believed that erythema nodosum is caused by toxins of tubercle bacilli.

Lupus vulgaris apparently is less frequent than it was in former years. Among approximately 7,000 children examined at the Lymanhurst School for Tuberculous Children, not one case has been discovered. However, the disease does exist among children. In diagnosis it has been shown that, when the superficial granulated tissue of ulcerative lupus is injected into guinea pigs, positive results are obtained in ninety per cent of the cases. This fact is also of great value in preventive work, as it proves that the disease is infectious. Wirz has reported sixty cases in which he used various methods of treatment. His best results were obtained by surgical excision, although he points out that this is not always feasible. Second to surgical excision, he found that x-ray therapy following iodine infiltration of the skin, is good. Heliotherapy has also been found of value in the treatment of lupus. A point to be borne in mind is that, in lupus, a high degree of allergy usually does not exist.

CERVICAL LYMPH NODES

Because they drain the greater part of the mouth, nose, throat, eyes and ears, tuberculosis of the cervical lymph nodes at one time was an extremely common condition among children. With the decreased incidence of tuberculous disease in the human family and the campaign for pasteurization of milk, there has come a great lowering of tuberculosis of the cervical lymph nodes among children. Usually this disease becomes manifest after the age of two years. In infancy it is uncommon to find tuberculous lymph nodes sufficiently large

to arrive at a diagnosis on clinical examination. Tuberculosis of the cervical nodes may be a primary disease. Bacilli taken into the nose, mouth and throat are carried by phagocytes directly to the lymph nodes, where they find lodgment, proliferate and cause disease. Usually it is secondary to an infection which appears somewhere about the mouth, nose, throat, or eye. Many cases of primary tuberculosis of the eye, tonsils, and so forth, have been reported, and, from the primary infection, just as is true in the chest, bacilli will quickly be carried to the regional lymph nodes. When the cervical lymph nodes become involved, there often is considerable enlargement of one node with less enlargement of those adjacent to it. There is little doubt that, in many cases, where tubercle bacilli reach the lymph nodes, they are brought under control without external manifestations. When the nodes do become definitely enlarged, they may, after a time, bring the disease under control and hold it in a state of latency for months, years, or the lifetime of the individual. Again, caseation may occur and eventually there is a rupture on the surface of the skin with suppuration over a long period of time. From such areas of disease, there may be no spread of tubercle bacilli so as to result in clinical disease elsewhere in the child's body but, in a fair percentage of cases, the disease appears in other parts. In nearly all cases there is spread to the hilum and tracheobronchial lymph nodes. Because of their accessibility, a diagnosis of tuberculosis of the cervical lymph nodes is not difficult. One must take into consideration the history of exposure to either human or bovine types of tubercle bacilli. Enlargements that have persisted over a period of three months or more, should have further study for tuberculosis. One must keep in mind that there are many causes of enlargements of the lymph nodes, such as carious teeth, diseased tonsils, acute upper respiratory infections, Hodgkin's disease and syphilis. Cutaneous tuberculin tests are of great importance. Some have advocated the use of subcutaneous tuberculin diagnostically, in order to bring about focal reactions. When this is done, the initial dose should

Fig. 1. A. S. Made from roentgenogram of neck of girl with extensive exposure to tuberculosis. Two definite areas of calcification in cervical nodes on right side and one on the left side.

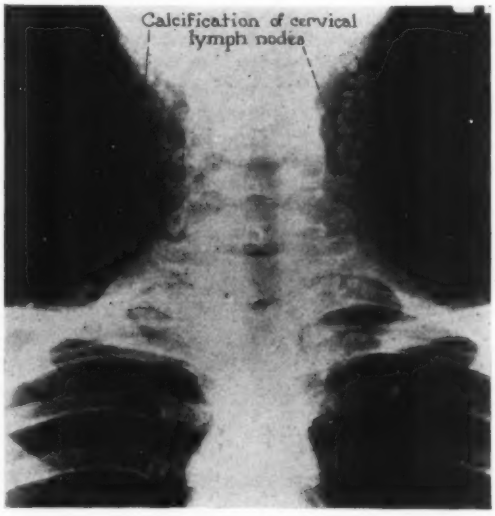

Fig. 2. M. E. Made from roentgenogram of the neck of girl with extensive exposure to tuberculosis and definite enlargements of the cervical nodes of both sides. Extensive calcifications are shown from the supraclavicular region and upward along each side of the neck.

never be greater than 0.5 mg. If the patient is highly allergic, a smaller amount, such as 0.1 m., will suffice for the initial dose. If no reaction of a constitutional or focal nature occurs, the dosage may be increased until a final dose of 5 mg. is given. When a focal reaction occurs, there will be an increase in the size of the lymph nodes involved. In the cervical region, as in the regions of the hilum, tracheobronchial and mesenteric nodes, the detection of calcification (Figs. 1 and 2) is excellent evidence of tuberculosis. Therefore, an x-ray examination of the cervical lymph nodes, particularly if the disease has existed for several months, may give one evidence that can not be obtained in any other manner. Biopsy may be resorted to, but it is preferable to arrive at a diagnosis without it, since surgery of the lymph nodes of the neck usually is not advisable. This procedure also is refused by many families. It should always be considered a last resort measure in diagnosis.

The general treatment consists of good diet, regulation of energy, expenditures as indicated in the individual case, and prevention of further exposure to tubercle bacilli. Heliotherapy is of value but one usually obtains better results by combining it with x-ray therapy. In extreme cases radium may be applied. Surgery usually is not necessary. However, in cases where one or more well walled-off nodes have been suppurating for a long time and have not responded to other forms of treatment, their complete removal is indicated. Again, with enlargements that have become densely fibroid or extensively calcified, which will result in permanent deformity, removal is indicated, but the old-time radical operation is almost never necessary and may even do harm.

The prognosis in cervical lymph node tuberculosis usually is good, but every case should be treated, for, otherwise, a certain percentage will later develop disease in other parts of the body, including the lungs.

When other lymph nodes, such as the axillary, cubital, and popliteal, become tuberculous, the diagnosis, treatment and prognosis are approximately the same as with cervical node involvement.

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CLINICAL SYMPTOMS IN THE PROGNOSIS OF TUBERCULOSIS

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(Read before the El Paso County Medical Society)

For the past century, the trend of the medical profession has been more and more to the study of the anatomy of the human body. Students are taught to know anatomy and to diagnose and treat diseases according to the anatomic pathology found. Physicians are inclined to give more importance to form and structure than to function. They are influenced more by evidence of pathology than by clinical symptoms. Anatomy and pathology have been given far more consideration than disturbance of function, in medical discussions. The demonstration of a specimen or microscopic slide showing deviation from normal, affords much satisfaction to the demonstrator and is received by the profession enthusiastically.

However, it is a fact that changes in function produce symptoms which are the physiologic pathology present and on which we must depend largely for our diagnosis. Furthermore, the restoration of normal function is the purpose of anatomy. So much consideration has been given anatomic pathology that we are prone to forget the physiologic pathology which really means so much in the diagnosis and treatment of disease. Clinical symptoms have not been given the consideration which they deserve. Accepting anatomic pathology as our basis for the diagnosis and treatment of disease has led to the grouping of specialists according to the organ involved. Consequently, if the same organism affects the throat, heart, lungs or appendix, we have the throat specialist, heart specialist, lung specialist or surgeon, respectively. Anatomically, pneumonia is confined to the lungs. Likewise, typhoid is confined to the intestines; yet pneumonia and typhoid are both general infections. Tuberculosis is also a general infection, and its intelligent study and treatment calls for an internist who has a broad view of not only the actual anatomic involvement but also of the clinical symptoms which indicate clearly the balance that exists between a patient's disease and his resistance.

Disease converts normal anatomy into pathology, but this would not be important if it caused no disturbance of function. Experienced tuberculosis workers know that the anatomic change may be very extensive, with little change in function, and not be serious. But marked disturbance of

function, with little or much pathology, is always serious. One cannot give a dependable prognosis from the anatomic concept. No physician would offer a prognosis based entirely on the anatomic pathology found. But the clinical symptoms representing the physiologic pathology or the patient's reaction to his disease, mean much toward a prognosis. Of course, a broader concept is obtained by a combination of both the anatomic and physiologic concept. Following the general trend to accept form and structure as being more important than disturbance of function, the classification of tuberculosis is based on the anatomic involvement, which is not nearly so important as the clinical symptoms.

Turban's classification of tuberculosis into three stages is the basis of all adopted classifications. It is based on the amount of involvement. It was his opinion (as stated by himself in discussing his classification before the International Tuberculosis Conference in Berlin, in 1908) that no other factor played such important part in the prognosis of tuberculosis as the extent or spread of the disease. The Turban Gerhardt classification adopted by the International Conference at Vienna in 1907, follows the same anatomic scheme suggested by Turban, but lessens the amount of involvement for the various stages.

Our National Tuberculosis Association followed the dominant anatomic basis but added a physiologic concept by requiring the consideration of the patient's symptoms in determining his status. This was real progress. But even in this classification, the anatomic entirely overshadows the physiological concept. The American Sanatorium Association, in 1916, adopted a classification further amplifying the importance of symptoms and permitting three classifications according to symptoms under each anatomic division, offering the following combination:

Incipient A, B, & C,
Moderately advanced A, B, & C,
For Advanced A, B, & C.

Turban offered his classification as a means of arriving at uniformity so as to enable physicians properly to arrive at more or less definite and dependable conclusions as to the results of treatment. He believed that involvement was the most important factor in the prognosis. He aimed at uniformity. But, in order to have uniformity, all examiners would have to examine with equal ability and equal care and would have to use equal judgment in interpretation. Such is not the case. Without the aid of the x-ray, the findings of different physicians in any case of tuberculosis vary considerably and even with aid of the x-ray, there is often a wide difference of opinion. The attempt at such a classification, however, has helped materially. It has demonstrated that the cases with lesser involvement without marked physiologic disturbance, are the more favorable cases for treatment. But it has further demonstrated, to more careful observers, that anatomic involvement, without consideration of symptoms, is a very poor basis for a prognosis. The physician attempting to follow these classifications is apt to have impressed on his mind that the extent of the lesion is the greatest factor and that the seriousness of the case can be mechanically measured. It causes him to concentrate on the extent of the disease instead of on the condition of the patient.

While the classifications used in America today are a combination of the extent and character of the lesion, on the anatomic side, and the clinical symptoms of the patient, on the physiologic side, nevertheless, the anatomic side remains uppermost in the minds of most observers. They attempt to assign mechanical exactness to processes which are by nature most inexact. While uniformity is the aim of all classifications, it is impossible in actual practice.

Strains of tubercle bacilli are of different virulence and patients have different resistance; therefore, it naturally follows that the balance existing between the disease and patient must differ. Likewise, the ability and methods of examiners differ. Many factors on the part of the patient must be considered, such as race, age, constitution and economic status. These and many other factors (all) differ in each patient. These variations make uniformity in disease unobtainable. The use of the x-ray has helped materially in determining the extent of the disease. But even the x-ray is not by any means definite, since the human element must enter into the technic of taking pictures and even more into the interpretation of the same. As the x-ray often reveals the inability of the internist to determine the character and extent of disease in the lungs, likewise the autopsy often exposes the faulty interpretation of x-ray pictures.

I should like to ask if anyone can arrive at mathematical exactness as to the involvement in a given lung by the aid of both physical examination and x-ray finding? Furthermore, if all the varying factors could be eliminated and a definite amount of anatomic involvement could be established, would

this give us a prognosis? After all, the ultimate reason for classification is to be able to determine the probable result in a given patient.

The divisions of first, second and third stage, or of minimal, moderately advanced and advanced, are purely arbitrary. They in no way tell us what is taking place. It is far better to have a whole lobe involved with the non-virulent fibroid type of tuberculosis than to have a small spot one inch in diameter necrosing. The first type of lesion shows that the fighting balance is on the side of the patient. Furthermore, a patient who withstands an extensive active lesion well has a far better chance to get well than one who withstands a small active lesion poorly. What patient and physician want to know, is not how much lung is involved, but how is the individual patient reacting to the disease that he has, and is he able to overcome it? Is the balance in favor of the patient or is it in favor of the disease? Are the normal smooth working cells of the body tissues disturbed and if so, to what extent? Whot is his physiologic as well as his anatomic response toward his tuberculosis? While we cannot and should not fail to consider the anatomic involvement, our chief consideration should be the amount of deviation from the normal physiological working of the body cells which has taken place. The relatively greater importance of the physiologic conception, as compared with the anatomic, is illustrated daily in our clinical experience. While, all else being equal, the greater the involvement the more serious the disease; yet, the extent of the lesion alone is but a minor point in destroying the patient. Furthermore, at best, it cannot be even approximately measured. The amount of disturbance to the normal working equilibrium of the body cells is the chief factor in prognosis—the physiologic rather than the anatomic deviation from normal. Disturbance of function is what brings the patient to the physician, and it is largely on this that the physician must base his dagnosis as well as his prognosis. The ultimate purpose of the physician and the ultimate hope of the patient is the restoration of normal function.

The physiologic pathology is more easily measured because it is expressed in symptoms.

One patient, after suffering a progressive necrosis destroying a large part of one or both lungs, is able to regain a fair degree of health because his physiologcii equilibrium is well preserved in spite of the extensive involvement. On the oteher hand, we sometimes see a small necrosing spot so disturb the physiologic balance of a patient and so depress his reacting power that the process spreads and becomes a menacing disease. We also sometimes see what seems to be a very slight anatomic lesion, accompanied by mild symptoms, and yet healed with the greatest difficulty. Such a case, in my opinion, is of mild virulence but fails to heal with usual rapidity because of the disturbance to the patient's physiologic balance.

In considering the relative value of symptoms one must consider their permanency and their effect on the recuperative power of the patient, as well as their cause. A temperature of 101, continuously, or at frequent and repeated intervals, is much more serious than one of only a few days' duration. It is also more serious if the patient is resting than if he is exercising. It is also more serious in a happy, optimistic patient than if maintained by worry. The rate of the pulse is also more important with the patient on complete rest over a long period of time and if taken carefully, eliminating the effects of exercise, excitement, worry, etc. Loss of weight is serious only when the cause cannot be corrected. Cough, expectoration and the number of bacilli are important only when considered over a long period of time. The same patient, studied when a small spot is necrosing, may raise much more, and examination of the sputum may show a great increase in the number of tubercle bacilli, and yet, in a few weeks the activity in the process may quiet down and the patient may be relatively free of cough and expectoration.

An analysis of all the symptoms of tuberculosis would show that the toxic symptoms are the ones which disturb the physiologic balance of the patient most. Certainly, the cause of death in tuberculosis is not due to the inability of the lungs to furnish oxygen. The cause of death seems to be a disturbance of the physiologic process to such an extent that the cells cannot continue to function in such a way as to keep the body in a state compatible with life.

The symptoms may be divided into three divisions: (1) reflex symptoms, (2) symptoms due to the process (per se) (3) toxic symptoms. Reflex symptoms are hoarseness, cough, digestive disturbances, (hypermotility and hypersecretion) circulatory disturbances, chest and shoulder pains and flushing of face. Symptoms due to the process per se are spitting of blood, sputum, frequent and protracted cold (tuberculous bronchitis) and pleurisy (tuberculous pleuritis). Toxic symptoms are malaise, fatigue, weakness, nervousness (digestive disturbance), increased

metabolic rate, loss of weight, increased pulse rate, night sweats and fever.

With this grouping, one can readily see that the reflex symptoms and those due to the disease process per se are not the ones which cause the serious changes in the body. It is the toxic group, or clinical symptoms, which really endanger life. The toxins of tuberculosis cause a more or less harmful stimulation of the nerve cells, producing a general nerve instability or nervousness. They also disturb the endocrine system, causing a loss of the chemical control of the body, with an increased acid condition of the tissues and with a diminution of the free water of the body, which interferes with the normal chemical activity of the body that should take place, and without which the body cannot be kept in a state of health.

Consequently, the prognosis in tuberculosis is individual. It does not depend on the extent or severity of the disease, but upon the manner in which the patient is able to preserve his physiologic equilibrium in spite of the extent and severity of the disease. Two patients may be suffering from tuberculosis of similar extent and intensity, as far as we are able to determine; and yet, one may sleep well, eat well, gain weight, and gradually lose his temperature, while the other sleeps poorly, is troubled with indigestion, loses weight and gradually grows worse. What is the difference? It is not the disease, but the manner in which the two patients react toward the disease. It is not the anatomic extent or the intensity of the involved area in the lung that makes the difference, but the difference in the individual physiologic equilibrium of the two patients.

The serious symptoms are always the toxic ones and are associated with clinical activity. Regardless of what symptoms may be most prominent, any patient who, for any great length of time, shows unfavorable reaction to the toxins, must be classed as unfavorable. In other words any patient with an increase of clinical symptoms after a prolonged period of bed rest, offers a serious prognosis.

The clinical symptoms, finally, are the key to the prognosis. The classification of tuberculosis into its anatomic stages has its place, but it is of minor importance as compared with the clinical symptoms.

The anatomic concept leads to a mechanical attitude toward the disease and leads away from the personal element, which is the more important factor of the disease. On the other hand, the physiologic concept leads to the personal element and encourages the study of each patient as a reacting organism. While a combination of both the anatomic and the physiologic concept is necessary to a clear, definite understanding of any case of tuberculosis, nevertheless, the prognosis depends chiefly, if not entirely, on the physiologic reaction of the patient, regardless of the extent or intensity of the disease.

CHRONIC MASTOIDITIS COMPLICATED BY RIGHT LATERAL SINUS AND JUGULAR BULB THROMBOSIS, MENINGITIS, AND BRAIN ABSCESS

JOHN J. McLOONE, M. D., F. A. C. S.
Phoenix, Ariz.

(Case report before the monthly staff meeting of St Joseph's Hospital at Phoenix, Oct 8, 1929)

L T., male, age 55, was admitted to St. Joseph's Hospital, August 8, 1929, referred by Dr. Christensen of Miami, Ariz., with diagnosis of probable sinus thrombosis.

Patient's present illness had begun ten days previously. Ever since boyhood, patient had frequent attacks of headache and pain in right ear, with intermittent purulent discharge. His family stated that present attack was more severe than usual and was accompanied by bloody vomitus, as well as intestinal hemorrhage. He also had severe dizzy spells before and during his present illness.

Examination:—General appearance showed a man extremely ill, mentality slightly clouded, but patient was rational. Fundi did not show any changes. Pupils reacted sluggishly to light.

Ear:—Right tympanum showed old anterior perforation. There was very slight tenderness generally distributed over the mastoid. There was a small amount of purulent discharge from middle ear which did not pulsate. There was no sagging of posterior-superior canal wall. Some pain was elicited when deep pressure was made in the region of the right internal jugular. Report on ear culture at this time showed some probable saprophytes and pneumococci.

X-ray and clinical findings showed extensive destruction of the right mastoid and a pansinusitis of same side. The spinal fluid was under slight pressure with no growth on culture. Cell count was in the neighborhood of 100. This varied, however, in the course of the disease, after five days reaching 4,000.

Temperature range on day of admission was 100 to 103½.

Dr. Raney saw the patient in consultation and made the following report.

"Heart rapid; moist rales at bases of lungs. Abdomen flaccid; not distended or indicative of any form of peritoneal irritation. Rectal examination: inspection normal; digital examination, probably a rectal polyp or small thrombosed internal hemorrhoid."

There was a distinct chill on evening of second day, followed by rapid rise of temperature to 105. A large amount of blood, partly dark but mostly fresh, was expelled by rectum. This had been preceded by abnormal distention and severe pain. A rectal polyp in this locality was not sufficient to

account for the large hemorrhage or for earlier bleeding.

The Widal was negative on the second day and was persistently negative throughout the illness. The temperature curve, especially before entering the hospital, general malaise, headache and hemorrhages, suggested a possible concomitant typhoid fever. The history of the ear condition, even in the absence of rapid pulse and positive blood culture, pointed to a diagnosis of sinus thrombosis.

He had been given a blood transfusion on the day of admission and several transfusions following operation. Transfusion was done as blood count and hemoglobin indicated. The beneficial result of blood transfusion in septic sinus thrombosis has been alluded to in one of my previous papers.

On the following morning operation was done under local anesthesia. Cortex was found to be thin. Lateral sinus was very superficial and placed far forward close to the external auditory canal. The external wall of sinus consisted of a large sloughing mass of tissue. The internal jugular was ligated above facial and below and resected. Several large degenerated and gangrenous clots were then removed from interior of sinus. Although bone over lateral sinus was removed beyond the knee there was no bleeding from above. There was partial bleeding obtained from below, but bleeding from above could not be obtained by any means whatsoever. At a later dressing, free bleeding from upper portion of sinus was obtained by inserting a catheter as far back as the torcular and applying gentle suction.

Hiccoughing, which was present from the time of admission and proved to be quite troublesome, showed some abatement following operation. For the first 24 hours after operaion, there was a slight improvement in the patient's general condition. After four days there was some rigidity of neck, partial bilateral Kernig's, but no positive Babinsky. Later facial twitchings were noted in the left eye and left side of face. Temperature ranged from 101 to 103. The average pulse rate was from 100 to 120, respirations quite high, in the neighborhood of 40 and 45. There was a gram-negative bacillus isolated from the spinal fluid which, when injected into a guinea pig, was found not to be pathogenic. Direct smear was negative. However, direct culture (that is from lateral sinus) showed some staphylococci and a few streptococci.

Patient was very restless and seemed to suffer pain referred to right frontal region. There was no paralysis of limbs on either side. There was no nystagmus. Lateral sinus wound, as well as region of jugular bulb, was satisfactory. Mastoid cavity had been completely cleaned and there was but a slight amount of drainage. Pus was noted, however. coming directly from upper region of sigmoid sinus. There had also been some purulent drainage from jugular bulb, but this had diminished.

On the 16th, spinal fluid count was reduced to 112 per c. mm. Copper reduction was present with an excess of globulin. Though sparse symptoms were present, the course of the case, especially spinal fluid findings, indicated a probable brain abscess.

Exploration for brain abscess was made. Operation was done without any anesthetic on account of patient being in coma. The cerabellum was first explored on account of its contiguity to the lateral sinus. After removing temporo-sphenoidal floor and excising dura, I found the whole lobe involved by large amount of degenerated brain tissue and thick pus. Bacteriological examination showed a staphylococcus. The abscess was encapsulated. Drains were placed anteriorly and posteriorly.

Following operation, patient's cnodition began immediately to improve. At 7 o'clock the same evening he had aroused from his coma and asked for water. His sensoria were not so cloudy and his general mentality improved even to the next morning. About three o'clock that afternoon, patient complained of a very severe and sharp pain in the lower lobe of left lung.

Examination disclosed a pneumonic process present, as well as the same condition on the right side, but not so marked. Old metastatic involvement of left lung was suspected and a diagnosis of multiple abscesses of same made. Early in the case, a metastatic involvement as the probable terminal factor was prognosticated. The case was a desperate one from the start, but indicated surgery offered the only possible hope.

This case illustrates the type of cerebral involvement, as well as systemic infection, that can result from a chronic mastoiditis. Middle ear infection of long standing, with repeated attacks of mastoid involvement, especially in the presence of cholesteatoma, should always be considered fraught with danger. This msatoid was, for a long period, the seat of a chronic suppuration which resulted in the destruction of its cellular elements. There were no structures remaining between the sinus and middle ear spaces. The right sinus plate was eroded, which gave sufficient evidence of the severely destructive power of the pathological process known as cholesteatoma.

The temporo-sphenoidal lobe abscess, as found in this case, is not at all an infrequent complication of a chronic neglected mastoiditis. Part of the systemic infection in this case expressed itself in metastatic abscesses of the lungs. Autopsy showed that these were numerous and of long standing and could be looked upon as the cause of the pneumonia, which, together with the general systemic infection, resulted in patient's death.

Antiustio.—The Council on Pharmacy and Chemistry reports that Antiustio is claimed by the manufacturer, Frederick Laboratory, Toledo, Ohio, to be "The Greatest Burn Remedy in Existence." A circular contains the following indefinite and nonquantitative statement of composition: "FORMULA Solvent Solution of Plumbic MATERIALS Subnitrate of Bismuth Zinc Sulphate and Iodide of Lead combined with mineral waxes." From this and other statements it would appear that Antiustio is a petroleum-paraffin mixture claimed to contain 5 per cent of bismuth subnitrate along with small but undeclared amounts of zinc sulphate and lead iodide. The Council found Antiustio unacceptable for New and Nonofficial Remedies because it is an unscientific preparation marketed with an inadequate statement of composition, under a name which is not descriptive of its composition and with claims that are exaggerated and unwarranted. (Jour A.M. A., November 16, 1929, p. 1559).

CASE OF DYSPNEA, CHEST PAIN AND SWELLING OF LEGS

(Case 15102, Case Records of Massachusetts General Hospital, from New Eng. Jour. of Med., March 7, 1929, p. 500.)

Case History.

An American iron moulder sixty-nine years old entered April 14 complaining of shortness of breath and swelling of the legs.

For fifty years he had had attacks of nocturnal dyspnea, at first associated with "severe colds in the chest" but for years coming without known cause, waking him up feeling suffocated and puffing as though under great exertion. The attacks lasted from half an hour to five hours. Five years before admission the shortness of breath came on insidiously, at first associated with exertion, then becoming progressively worse until even walking brought it on. For four years he had urinated twice at night. Two years before admission at the end of a day's work he became dizzy and short of breath, had some dull precordial pain and was unconscious for half an hour. Then he recovered without treatment and went home unassisted. Since that time he had not worked. He had felt similar attacks coming on, but rest seemed to prevent their developing. For four months his chronic cough had been more frequent and precipitated by exertion.

His father died of gastric cancer.

Since he was nineteen the patient had had "bronchitis"—productive cough except for about three months each year usually in summer. At twenty-five he had Neisser infection. Since the fainting episode two years before admission his memory had been steadily failing. Two years before admission his weight was 235 pounds, six months ago 223 pounds. He had been losing weight for the past year.

Clinical examination showed an obese, cyanotic, dyspneic, wheezing old man with dehydrated, almost icteroid skin. Teeth, old carious snags. Barrel chest. Lungs full of piping squeaks, inspiratory and expiratory rales. Apex impulse of the heart not found. Percussion measurements: left border 15 centimeters from midsternum, 7 centimeters outside the midclavicular line, right border 1.5 centimeters to the right, supracardiac dullness 6 centimeters. Action absolutely irregular. Fibrillation. A rough systolic murmur heard all over the precordia and into the neck. No diastolic heard, although on account of the rapid heart action and fibrillation the examination was not satisfactory. There was pulse deficit. Pulses not synchronous. Artery walls thickened and tortuous. Blood pressure 190/100. Liver four fingerbreadths below the costal margin, tender, edge felt. The legs showed massive pitting edema, the sacrum moderate edema.

Urine and blood not recorded. Renal function 50 per cent. Non-protein nitrogen 30 milligrams. Wassermann not recorded.

Temperature 98.6° to 103.5° rectal, pulse 80 to 108, respirations 55 to 35.

The patient was given the equivalent of 15 grains of digitalis intramuscularly within twelve hours after admission, but when seen during the night was having violent dyspnea and orthopnea and could not be quieted with morphia. The rate was regular.

The morning after admission the temperature rose to 103.5°. An electrocardiogram showed auriculoventricular dissociation, auricular rate 80, ventricular 90, irregular; left bundle branch block, varying; few more normal beats in lead I. Another electrocardiogram showed idioventricular rhythm with variable intraventricular block; no evidence of auricular action; rate 50, almost regular. The patient died at the end of the electrocardiogram.

The following discussions of this case were given before the Yavapai County Medical Society at their regular meeting of October 29th:-

DR. BAYARD SULLIVAN

The pulmonary phase of this case presents a picture that is confused or overlapping. This patient came to the hospital an obese, cyanotic, dyspneic, wheezing old man, with barrel shaped chest, piping squeaks in chest, inspiratory and expiratory rales. The history says he was an iron moulder by trade and had dyspnea and shortness of breath for fifty years. This is a fairly definite history of pneumoconiosis, chronic bronchitis, bronchal asthma, pulmonary tuberculosis (fbiroid type), or emphysema. We believe these symptoms more definitely describe pneumoconiosis than any of the others mentioned.

We know that pneumoconiosis is due to the inhalation of dust, hard and gritty. It is divided into several groups: anthracosis due to the inhalation of hard coal, siderosis, and silicosis or chalicosis. It is also divided into two general classes,—organic and inorganic dust, but as a rule inorganic dust plays the chief role in cases of pneumoconiosis. The dust inhaled is usually of silica content and the greatest damage is due to sharp edges and cutting.

The authorities I have read on pneumoconiosis are Kober and Hanson on Occupational Diseases, Mock on Industrial Diseases and a chapter in Tice Practice of Medicine.

The pathology of pneumoconiosis is chiefly that of fibrosis of the lungs. The dust inhaled is carried to the alveoli of the lungs and, through the phagocytes of the body, nature attempts to overcome the foreign matter. A serous discharge deposits and fills up the alvoli of the lungs and fibroses there. Fibroblasts of endothelial cells may be thrown out.

The chief symptom of pneumoconiosis is shortness of breath. Some patients may have a large amount of pneumoconiosis and few symptoms, and others a small amount and many symptoms. This dyspnea is, of course, due to the obstruction of alveoli of the lungs. In some cases there may be fibrotic pleurisy with spitting of blood.

Chronic bronchitis is one of the earliest phases of pneumoconiosis. In this case the history gives bronchitis at the age of nineteen.

We have no x-ray report and x-ray is one of the chief diagnostic helps, showing the mottled lungs. Fibrous mottling is distributed rather generally.

In bronchiestasis the chief symptom is the amount of sputum. In this case the history mentions only a productive cough.

One of the effects of pneumoconiosis is the strain on the right heart. Usually the right heart has an extra load to put the blood through the lungs and finally fails.

The five things mentioned must all be considered, —chronic bronchitis, chronic bronchial asthma, bronchiectasis, tuberculosis and emphysema, as well as pneumoconiosis. The terminal condition we believe is hypostatic pneumonia and passive congestion.

DR. B. L. JONES

We have the case of a man sixty-nine years old who worked up to the age of sixty-seven, practically

to the end of his life. He has broken down completely in the past two years, and has a history of dyspnea for the past fifty years. There are eighty uncommon causes of dyspnea and about twenty common causes.

This man had marked edema of the legs and of the sacrum; had evidence of enlarged heart by percussion, and evidence of myocardial disease according to the electrocardiogram. According to clinical examination he had an irregular heart; the history says he had fibrillation. So we have the end picture of a man with congestive heart failure. He had at that time arteriosclerosis, clinical examination showing tortuous arteries. He had dyspnea over many years, which we believe is due to pneumoconiosis, and finally also cardiac failure.

This man had a breakdown two years ago, with sudden loss of consciousness, some dyspnea, dizziness, shortness of breath and dull precordial pain. What happened at that time? We must consider the great types of heart disease; rheumatic heart disease syphilitic heart disease, arteriosclerotic heart disease, coronary disease, and after that congenital heart disease. We can eliminate rheumatic heart disease because there is nothing in the history to point to it. We can eliminate syphilitic heart disease because there is nothing in the history or clinical examination to point to it, further than that this heart block they speak of could be caused by gumma involving the conductive system of the heart; but we do not believe that gumma is the cause in this case. Hypertensive heart disease with arteriosclerosis, general, and possibly coronary disease with thrombosis and infarction are less easily eliminated. Either could have caused the attack of two years ago, with rupture of a vessel of the brain, giving short loss of consciousness and recovery. But we do not believe death was caused by rupture of a cerebral artery.

The history states this man had marked dyspnea at night. Paroxysmal dyspnea may be due to a non cardiac conditions or due to cardiac. In the non cardiac we think of acidosis. Myocardial infarction may occur in the absence of any pain but with sudden onset of urgent dyspnea.

There are two conditions which may occur in advanced heart disease:- cardiac asthma and acute pulmonary edema. Many physicians do not take very seriously the question of cardiac asthma, but there is a definite type which does occur in advanced heart disease. It comes on when a laboring left ventricle tries to meet the demand put upon it, and tries to supply the necessary oxygen to the respiratory centre. At the same time that the respiratory centre is calling for more oxygen, the peripheral circulation begins to contract to force the blood into the organs that call for more oxygen to the respiratory centre. Secondly, we have acute pulmonary edema, with which you are all acquainted.

We know that there was definitely heart-block in this case, which was determined by the electrocardiogram. Heart-block, involving the conductive system of the heart, may be caused by congenital difficulties, toxic or inflammatory conditions, severe anemia, acute rheumatism, syphilis, subacute septic endocarditis, vascular lesions with infarct of the myocardium, and, in old people, obliterative processes in the arterioles. The last mentioned is a common cause in old people and we believe it occurred in this case. Rarely, we may have tumor of the neck causing depression of the conductivity of the bundle of His, due to pressure on the vagus. Also drugs such as the digitalis group depress the conductivity of the bundle of His.

There is only percussion in this case to determine the size of the heart. That is an extremely large area of dullness and makes one think of pericardial effusion. We have not gone so far as to make a diagnosis of pericarditis in this case, but it could very easily occur in a man so edematous.

We believe there is artiosclerotic heart disease, emphysema with pneumoconiosis which has gone on for years. What caused death? There are two probable causes, cardiac infarction with rupture of the heart, or fibrillation of the ventricles. So we have as our diagnosis: Pneumoconiosis; hypertensive heart disease; arteriosclerosis, general; congestive heart failure; emphysema pulmonary and probable cardiac infarction causing death.

The following discussions of this case were given before the Clinical Club of Phoenix, at their regular weekly meeting of Nov. 18th.

DR. FRED G. HOLMES

This is evidently a case with rather long standing heart disease gradually failing, with hypertension, arteriosclerosis, passive congestion, the terminal factor probably being a bronchopneumonia.

Evidently his nocturnal dyspnea attacks were associated with the so-called bronchitis attacks as they started at the same time. This bronchitis was most likely a bronchiestasis with a productive cough lasting fifty years. However, the nocturnal dyspnea attacks in later years took on the characteristic symptoms of a failing left heart which later began to show symptoms during the day with shortness of breath on exertion. During this time there was probably an advancing arteriosclerosis with consequent kidney damage, hypertension and loss of mental acumen.

Two years before admission, after exertion he had what was perhaps a circulatory disturbance due to his arteriosclerosis, with pain over the heart, dizziness and unconsciousness. There may have been a slight coronary occlusion.

The terminal picture is that of a hypertrophied and dilated heart, an enlarged aorta probably with atheromatous patches and sclerosed coronary arteries and a generalized arteriosclerosis of the peripheral arteries, brain, kidney, etc. Emphysema and bronchopneumonia. If a bronchopneumonia did not exist, it is possible that he had a terminal pericarditis.

The electrocardiogram shows a profound disturbance in the conduction system of the heart, there being not only a complete block in the bundle of His but also a left bundle branch block and variable intraventricular block. This was probably due to the arteriosclerosis.

There is apparently no definite evidence whether an endocarditis existed or not.

Diagnosis:

Hypertrophy and dilation of the heart.
Arteriosclerosis.
Hypertension.
Passive congestion.
Emphysema.
Bronchiestasis.

DR. VICTOR RANDOLPH

Nocturnal dyspnea associated with severe "colds" in the chest, and later without any such "colds," lasting over a period of fifty years predicates bronchiectasis. This would occur as a result of continued "colds" and asthmatic attacks even if it were not the cause of it, so we know that this patient has chronic bronchiectasis. The attacks during the past five years make one think of cardiac failure in addition. The fainting episode two years ago must have had cardiac cause. Physical examination confirms the findings thus far and adds the diagnosis

of auricular fibrillation. There is also arteriosclerosis both general and renal. The electrocardiogram would indicate diffuse lesions in the myocardium were it not taken so shortly before death. Diagnosis: Bronchiectasis, arteriosclerosis, cardiac failure, hypertension.

The following discussions of this case were given at the Clinico-Pathological Conference at the Massachusetts General Hospital:-

DR. RICHARD C. CABOT

Taking the facts stated in the opening sentence and no more, you make a guess that that is the arteriosclerotic hypertensive type of heart disease.

The first sentence in the history of the present illness sounds like asthma.

At the end of the past history I still have the same impression that I had in the beginning.

NOTES ON THE PHYSICAL EXAMINATION

"Almost icteroid skin." I think the examination ought to result in a little more definite a statement than that. Did the patient have any jaundice post mortem?

Dr. Tracy B. Mallory: No.

Dr. Cabot: The physical examination runs perfectly to type so far.

The urine and blood are not recorded, presumably because he died so quickly.

The non-protein nitrogen is 30 milligrams; so far as that goes we have no good reason to suspect the kidney.

The heart rate was regular, so it seems as if the digitalis had taken hold.

The patient died during the electrocardiographic examination, which shows that examination is not always the best thing for people even when it is good for the diagnosis.

DIFFERENTIAL DIAGNOSIS

I do not see anything essentially different to say from what we started with. He has heart disease, presumably of the hypertensive arteriosclerotic type. He has also had angina for a long time. We have nothing to refer us to any other organ. The question remains, did he have a fibrous myocarditis? I think it is probable, though I do not think there is anything in the electrocardiogram that would absolutely prove it. In a person with such lesions as he has at his age myocarditis is probable. So I think Dr. Mallory will tell us he has hypertrophy and dilatation without valve lesion and general passive congestion of all the organs, arteriosclesosis probably, and that is all.

A Student: Are those peculiar rhythms always a sign of myocardial failure? Don't you make the diagnosis of myocarditis on the basis of peculiar rhythm?

Dr. Cabot: No. I think we ought to make a distinction between myocardial failure and myocarditis, which ought to mean a fibrous process as a result of old infection. Such electrocardiographic changes suggest but do not prove myocarditis.

Don't you find some changes in the heart muscles to account for the disturbed rhythm?

Dr. Mallory: Undoubtedly the conduction processes can be disturbed without any discoverable anatomic basis.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Hypertensive and arteriosclerotic heart disease, with congestive failure.
Auricular fibrillation.
Bronchopneumonia.

DR. RICHARD C. CABOT'S DIAGNOSIS

Hypertensive and arteriosclerotic heart disease.
Hypertrophy and dilatation.
Arteriosclerosis.
Possibly fibrous myocarditis.
Chronic passive congestion, general.

DR. TRACY B. MALLORY

ANATOMIC DIAGNOSES

1. Primary disease.
   (Hypertensive heart disease.)
   Hpertrophy and dilatation of the heart.
   Arteriosclerosis.
2. Secondary or terminal lesions.
   Emphysema.
   Anthracosis.
   Chronic passive congestion, general.
3. Historical landmark.
   Cholelithiasis.

Necropsy showed two important lesions which are undoubtedly directly connected with his death. One was emphysema of the lungs, which incidentally also showed unusually marked anthracosis, and the other was cardiac hypertrophy with dilatation of the cavities. The heart weighed over 600 grams and showed marked hypertrophy of both ventricles. The right ventricle wall measured 10 millimeters, an increase over normal of almost 300 per cent, and the left measured 25 millimeters, approximately twice normal. There was a moderate degree only of chronic passive congestion shown in the liver and spleen. This fits fairly well with the great hyperthrophy of the right heart, suggesting that emphysema had much to do with his condition, since it is well known that passive congestion of the liver is not the rule in emphysema. We must, however, also assume a hypertension in view of the hypertrophy of the left heart and the hyalin sclerosis of small arteries found in the kidneys. These did not, however, show anything which could fairly be described as a genuine vascular nephritis. Microscopic examination of the heart failed to show any increase in fibrous tissue.

An incidental finding was a small and greatly scarred gall-bladder containing faceted stones.

NOTE BY DR. CABOT

The case should be remembered as proof that extensive disturbances of conduction—as shown by the electrocardiogram— may exist without discoverable myocarditis, macroscopic or microscopic.

CASE OF DYSPNEA, WEAKNESS AND EDEMA.

(Case 14431, Case Records of Massachusetts General Hospital, from New Eng. Jour. of Med., Dec. 13, 1928, p. 1217).

Case History.

First Admission. A German-American salesman sixty years old entered January 24 complaining of dyspnea, weakness and edema.

His illness had come on during five years of unsuccessful business and increasing worry. For a year he had not felt well. He had however had no definite symptoms until late in the last September. Then he had severe and rapidly increasing dyspnea, which became incapacitating. From October 1 to January 22 he was in a Christian Science sanatorium, where he never had medication or complete rest in bed. After the first week he was confined to his room until shortly before he left. Early in October he was seized with dropsy, which became very severe. He had orthopnea, and occasionally urinated once at night. In the latter part of De-

cember and in January the edema became less. He had been hoarse for some time and occasionally had sputum. January 23, the day after his discharge, he was suddenly seized in the night with respiratory distress, weakness and a vague thoracic discomfort bordering on pain. The morning of damission he thought his sputum contained blood.

His father died of Bright's disease.

The patient had been subject all his life to nosebleeds, occurring every day for five to ten days at intervals of two to five years. The last attack was three months before admission. In his youth he had asevere headaches. His only serious illness was a left renal or ureteral stone. At twenty-five he had a fairly severe attack of breathlessness after shoveling snow. In the past ten years he had three or four attacks of palpitation following severe exertion. Recently he had some left tinnitus. He denied venereal disease but not exposure.

Clinical examination showed a fairly well nourished man lying propped up in bed, restless and breathing rapidly. The subcutaneous tissues from the waist down showed brawny edema. Pyorrhea. Tongue dry. Tonsils very large. Pharynx injected. Barrel chest, with apparently limited expansion. Apex impulse of the heart not seen or felt. Left border of dullness 9.5 centimeters to the left of midsternum, half a centimeter outside the mid-clavicular line. Right border 3.5 centimeters. supracardiac dullness 6 centimeters. Action regular, rate 90. Heart sounds heard very well in the axilla. No second sound at the apex. Aortic second sound barely heard. A harsh musical systolic murmur heard everywhere. The murmur and the sounds in the pulmonic area seemed disassociated. Radials and branchials hard, branchials very tortuous. Blood pressure 155/100 to 170/90. Electrocardiogram: normal rythm, rate 75, change in pacemaker in Lead II, varying shape of P wave. Lungs: coarse crepitant rales in left axilla, medium crepitant rales in lower right back to the level of fourth rib; in left lower back to sixth rib dullness to flatness, diminished tactile fremitus, breath sounds, whispered and spoken voice, coarse crepitant rales. No egophony. Examination of abdomen obscured by subcutaneous edema and voluntary spasm. Dullness below right costal margin. Scrotum and penis showed edema, the scrotum a large crusted ulceration due to irritation. Very hard edema of legs and ankles; skin red, warm and scaling. Hyperesthesia of soles of feet. Motion of knees limited. Pupils somewhat small (morphia?), reacted only slightly to distance, not at all to light. Knee-jerks and ankle jerks normal. Arteries of both fundi slightly tortuous.

Urine: amount normal except 75 to 85 ounces January 17 and 28, specific gravity 1.008 to 1.024, a trace to a slight trace of albumin at two of five examinations, rare leukocytes at two, rare red cells at one. Renal function 15 to 25 percent. Blood: 19,400 to 10,800 leukocytes, 71 per cent polymorphonuclears, hemoglobin 90 to 75 per cent, reds 4,700,000 to 5,304,000, anisocytosis with a tendency to macrocytosis and occasional basophilia and tailed forms, platelets slightly reduced. One Wassermann moderately positive, one negative. Hinton negative. Non-protein nitrogen 39.

Temperature 100° by rectum to 97° by mouth. Pulse 100 to 68, with a deficit of 2 to 7 beats January 27 to 30. Respirations normal.

By x-ray the heart shadow appeared large and was prominent both to the right and the left. The aorta was prominent in the region of the knob. Both lung fields were less radiant than normal, especially at the bases. The outline of the left diaphragm was obliterated in part, perhaps by the heart shadow and motion. The larger lung markings were particularly prominent.

January 25 the systolic murmur was loud, squeaking, transmitted to the axilla. There was a questionable systolic, also squeaking and at the same pitch, over the tricuspid area. Next day there was effusion at the left base. During the next few days he did very well, lost 20 pounds, and by January 29 had no edema of the legs, abdominal wall or scrotum. February 1 he suddenly became irrational. The next day he was mentally clear again, and after this felt well except for occasional back pain. When he sat up long he felt tired and dyspneic. His urine seemed a little warmer than usual. February 7 there was dullness with bronchial breathing and increased spoken and whispered voice at both apices, marked over the right posteriorly; egophony was questionable here, positive on the left.

February 8 he was discharged improved.

History of Interval: On leaving the hospital he stayed for three weeks in convalescent homes and a hospital, and kept fairly quiet. Then he went home, and from that time did a good deal of walking and stair climbing. He used two pillows at night. Toward the end of March he began to have swelling of the legs and dyspnea, sittiug up most of the night. These symptoms grew progressively worse. About the middle of April his abdomen began to swell, and perhaps a week later his penis and scrotum, interfering with the passage of urine and causing pain on starting the stream. For two weeks he had been in bed. His physician increased digitalis to two grains a day and three grains every other day and gave him a tenth of a grain of morphia with atropin at night. For a wek his condition had been about the same.

Second Admission: April 28, eleven weeks after his discharge.

Clinical examination was as before except as noted. He had marked Cheyne-Stokes respiration with gasping hyperpneic phase. His lips were somewhat cyanotic. The skin was dry, inelastic; over the legs tense and thickened, somewhat scaling. There were coarse moist rales at the base of the right lung and possible fluid at the left base,—dullness with absent breath sounds and fremitus. The cardiac impulse was strong. The apex was seen and felt in the fifth space. The midclavicular line is now recorded at 8.25 centimeters from mid-sternum, the left border of dullness 5.5, right border 3, supracardiac dullness 5.5. The sounds were of poor quality, the first sound obscured by a loud high pitched systolic murmur. There was a questionable systolic thrill at the apex. No diastolic murmur was heard. There was no pulse deficit. The blood pressure was 190/130. The sounds were of uniform quality. There were signs of ascites. The abdominal wall was tense, tympanitic over the center. There was tense hard edema of the entire lower legs extending up over the lumbosacral area and back. The right lower arm showed pitting edema. The rest of the examination was postponed.

Urine not recorded. Blood: 13,750 leukocytes, 84 per cent polymorphonuclears, hemoglobin 80 per cent, red count and smear normal.

Temperature 90° to 104.1°. Pulse 80 to 108. Respirations 10 to 30.

The patient was given digitalis intramuscularly and morphia 1/6 grain every three hours. Venesection was postponed because of the absence of marked cyanosis and edema of the lungs. He remained in about the same condition until early the morning after admission. Then he had tracheal rales, restlessness and increased dyspnea. Morphia was pushed and he was given scopolamin 1/200 grain. 250 cubic centimeters of blood was with-

drawn. During the venesection the blood pressure began to drop; within half an hour it fell from 200/135 to 138/88. He failed rapidly and at midday died.

The following discussions of his case were given before the Yavapai County Medical Society, at their meeting of October 29th:-

DR. GEORGE S. McCARTY

The picture presented in this case is of an old man of sixty years who, for at least five years previous to admission to hospital, was conscious of impending trouble, but refused to consent to proper medical treatment, rather pinning his faith to Christian Science until marked disturbance in health appeared.

The history gives a detailed account of his previous condition, increasing respiratory distress, dyspnea, dropsy, urinating at night, frequent nose bleed, occasional sputum (thought to be streaked with blood), severe headaches, and frequent palpitation which lead one to suspect a condition of the heart or kidney or both, as initial cause.

Clinical examination shows a man very restless and gasping for breath, with subcutaneous edema. There is enlargement of the heart, as shown by examination, and a harsh musical systolic murmur heard everywhere. The blood pressure is high. There is tonsillitis and pyorrhea. If we were looking for a focus of infection to account for the heart lesion, it would likely be found here. A slightly positive Wassermann and questionable venereal history suggest lues. The urine in this case indicates decreased kidney function,—there is only a trace of albumin, rare leukocytes and occasional red cells, no casts. We are inclined to think this is not a marked abnormality but rather indicates a decompensating heart.

This patient was hospitalized from January 24th to February 28th. During that time he made some improvement and was discharged as improved, whether or not of his own accord we can not determine. April 28th he was re-hospitalized. Physical examination was the same as the previous one, with the exceptions noted,—Cheyne-Stokes respiration, cyanosis more marked, moist rales in bases of lungs, dullness and fremitus,—which would lead us to believe there is fluid in the base of the lung. It is noted that he was given digitalis, morphine, scopalomin, in heroic doses without response, and finally venesection was done. The blood pressure dropped very rapidly, patient collapsed and died.

Our diagnosis is arteriosclerosis, from the fact that his brachial and radial arteries are extremely tortuous; possibility of syphilis on account of enlargement of the heart and aorta and questionable Wassermann; cardiac hypertension, which was followed by decompensation; possibly myocarditis followed by pulmonary congestion; and, terminally, broncho-pneumonia.

DR. C. C. BENEDICT

Looking over this history for a focus of infection, for we believe this condition may be due to absorption, the only lead given is pyorrhea. It is possible that a man in such a serious condition would not give information of what he considered some trivial infection, such as sore throat or rheumatic trouble. At any rate we can find only pyorrhea as such a focus.

It is quite evident to us that this man had a decompensating heart. The evidence points in that direction. In the first place, there was edema, which began in October (before admission in January); this edema began in the most pendant parts and gradually ascended; also moisture at both bases, which we take to be due to backing up and failure of right heart. There is also dyspnea and cyanosis, although shortness of breath was not particularly marked; he complained of it at times from the beginning, in fact at the age of twenty-five we are told he had dyspnea on exertion. This edema also fluctuated considerably. For instance, it is noted on first examination that the apex beat was not seen or felt, but on second examination it was both seen and felt in the fifth interspace. Also the left border of dullness at first examination was 9.5 centimeters to left of midsternum, and 10.5 at second. While there is not much value attached to percussion for cardiac dullness, neither is it of much value as a method of outlining the heart, because examination or percussion of the heart by the same examiner on two successive days may show a wider difference than shown here. As to the murmur,—at first examination it was harsh and musical and heard all over the precordium, at second examination it was called "loud" pitched. My understanding of pitch is high or low. I do not know what "loud" means. Also at that examination it was freely felt at the apex.

I want to refer to examination of a series of 318 cases by Berger in 1927, of murmurs of questionable origin. He classified murmurs under these heads; the asthenic habitus group, one group due to transverse position, one group due to displacement and distortion, and the group which proves to be organic. The value of a murmur depends much upon the position of the patient and upon the relative size of the heart as compared to the size of the individual and the shape of the chest. This man had a barrel shaped chest, and while we believe there was an organic murmur, it is possible it may have been due to the position of the heart in the chest and its capacity. The heart may have been in some suspended position, which will produce a murmur without valvular difficulty, by reason of the blood being thrown in a crooked direction. In other words, the blood stream makes a change in direction, producing a sound resembling a murmur. This may also occur in ascites. It has been noticed when the ascites disappears the murmur will also disappear.

The blood pressure on first examination was 155/110 to 170/90. It is well known that a high diastolic pressure argues for cardiac collapse and as we go on we will show the significance of this high diastolic pressure. Second examination, 170/90 pulse pressure of 80 which is very high; third 190/130 and later, just before death, 200/135, pulse pressure 65. After venesection it dropped to 138/88, which we believe is due to impending death and not necessarily to the withdrawal of 250 c.c. of blood. The diastolic pressure of 135, we believe, is very significant. Practically all authorities agree that a diastolic pressure that high means hypertension; 160 systolic or over constitutes hypertension.

While the electrocardiogram report in this case is not of particular moment, it is believed it does indicate some involvement of the myocardium in this respect, quoting from the history, "change in pacemaker in Lead II, varying shape of P wave." Lead II is taken from the right hand and right foot and variation in the P wave will be due to different things. There is a change in the P wave due to change in location of the impulse stimulus, or change in the mass of the myocardium. This unequal distribution means that the muscle is thicker in one place and thinner in another, resulting in interruption of stimulus as it travels, and through obstruction to flow of impulse, which may be due to

change in structure of the impulse transmission paths.

The pulse deficit of 2 to 7 is an indication of some involvement of the myocardium. Broadbent said that the functional capacity of the heart depends primarily uponmu sculature, yet the efficiency of the heart as a pump may be impaired to a great extent by disease of the valves or by disorder of rhythm. So that musculature is not necessarily the only factor concerned with irregular heart and failure. According to that, impaired function may be classified as valvular disease, myocardial disease or diseases of rhythm. Valvular disease of severity produces changes in the walls and cavities, hypertrophy of the ventricular wall and dilatation of the other cavities, in the effort made to compensate. From the degree of these changes then, you may estimate the extent of the lesion, and from the effect of exercise on the heart you may judge the efficiency of the compensation. To estimate the extent of myocardial change is more difficult. The characteristics of the cardiac muscle are contractility and tonicity and have much to do with functional integrity. Conductivity, excitability and stimulus production have more to do with disorders of rhythm and are not much concerned in our case. Contractility is that property of heart muscle that enables a ventricle to completely empty itself within a reasonable time. A ventricle that can not empty itself loses its tonicity. Tonicity maintains the tone,—it must completely empty itself and be ready at all times to respond to stimulus, produce contraction, so that the ventricle does not become overdistended. It is the loss of this function that figures largely in cardiac failure. The main symptom of loss of contractility is pain on exertion, and when that pain on exertion is unaccompanied by ty. The measurements show this heart was encharacteristic valvular findings, it is angina.

In this case we find evidence of valvular difficullarged. Summing up the findings then, it is our belief this man had valvular heart disease with decompensation, that he had arteriosclerosis, secondary edema, and broncho-pneumonia. It is possible too, that he had myocarditis because, taking for its face value the electrocardiograph reading, it is believed that only in diseases of the myocardium could that pulse deficit be found.

The following discussions of this case were given before the Clinical Club of Phoenix, at their regular weekly meeting:-

DR. J. M. GREER

The first thing of course that strikes our attention in this case is the dyspnea and edema, coming on, although rather rapidly, yet over a period of time and not following any acute condition of disease. This suggests immediately a cardio-renal condition, or disease. However if we wish to be more complete and thorough in our differentiation we should probably consider, and rule out, if possible some of the other things that may cause dyspnea and edema, such as pulmonary and hepatic conditions for example. The age of patient must be kept in mind as well as his occupation and unsuccessful business ventures, as these all predispose to cardiorenal syndrome or perhaps more properly a neuro-circulatory syndrome.

The dropsy spoken of early in October probably meant a failing compensation of the circulatory system. Orthopnea is seen in several conditions but is, of course, common in failing cardiac compensation. The hoarseness and sputum could be accounted for by passive congestion. The sudden seizure of respiratory distress, weakness and thoracic discomfort, point toward a mwocardial weakening. The blood tinged sputum is not infrequent in myocardial disease. The family history of Brights disease is, of course, to be noted but to me means very little unless we know more about the history of the disease in the parent. The history of nose bleed may point to an inherent weakness in the arterial system. The history of palpitation means to me a cardiovascular weakness of some sort. However, we should know more of the habits of the individual to be at all sure of this. The tinnitis complained of no doubt means a degenerative process in the peripheral vessels.

With the above suggestions received from a consideration of the history we now come to the physical examination.

He is propped up in bed, respiratory difficulty, suggesting trouble primarily with heart, lungs, or kidneys. The restlessness probably points more towards the heart. The dependent edema suggests circulatory failure rather than a kidney condition. The barrel shaped chest suggests a chronic condition extending over a long or relatively long period of time. The fact that the apex impulse is not seen or felt although the edema only extends to the waist immediately suggests a heart condition either inherent in the heart itself or fluid in the pericardial sac. The increased area of dullness indicates an enlargement of the heart or fluid in the pericardium. The weak heart sounds suggest a general weakening of the heart itself and their being heard in the axilla probably means a dilatation. The varying shape of the P wave in the electrocardiogram probably means an erratic heart muscle and one that responds abnormally to the natural stimuli. Dr. Randolph may be able to enlighten us more on this point. The lung findings are consistent with a passive congestion.

The thing suggestive to me in the blood pressure, in the presence of hard radial and brachial arteries is that with a relatively normal heart muscle one would expect a much higher pressure. Therefore, this would indicate that we probably are dealing with a heart muscle that is diseased. The pupillary findings are only suggestive of a luetic etiology. The urinary findings seem to me to take our thoughts away from a renal condition and point again to a heart condition. The blood picture does not mean much to me but maybe some of you can point out something of value in it. The Wassermann of course is only suggestive. The x-ray findings again bring our thoughts back to the heart.

The improvement in the patient after January 25, was, no doubt, due to rest and perhaps treatment or management. The irrational condition on February first can be accounted for and suggests an atheromatous condition of the cerebral vessels. The tired and dyspneic condition of the patient after sitting up again suggests cardiac insufficiency. Just what the warm urine means I do not know. May be some of you can enlighten us upon that subject. The return of dyspnea and edema again after leaving the hospital after walking and climbing stairs again bring out thoughts around and suggest cardiac incompetency.

His second admission:

The Cheyene-Stokes respiration, cyanosis, edema and chest findings with cardiac dullness increased systolic murmur, strong impulse with ascites and edema indicate a heart that is trying to do its work but unable to do so. The leukocytosis as well as temperature curve may be accounted for by congestion due to circulatory failure. Death no doubt came about through circulatory failure associated with pulmonary edema and congestion.

Diagnosis: Myocarditis, passive pulmonary con-

gestion, arterial sclerosis, with degenerative changes in the liver and kidney.

DR. FRANK J. MILLOY:- Arteriosclerosis as primary factor.

DR. VICTOR RANDOLPH:- Arterio-renal disease, with cardiac hypertrophy and failure as secondary.

The following discussions of this case were given at the Clinico-Pathological Conference at the Massachusetts General Hospital:-

DR. RICHARD C. CABOT

It is noticeable how often histories begin as this does. It is the beginning of a larger number of cases than any other. One might say "Why do they hand out only cardiovascular cases here?" The answer is, cardiovascular renal disease is the commonest cause of death, a group as big as any other two groups. Including nephritis, cerebral hemorrhage and the various kind of heart disease it is more than twice as common as any single cause of death that we can name. So it is not surprising that it occurs so often here.

This is a perfectly straight cardiovascular history. If nothing comes out to the contrary we should say this man has some form of heart and vessel trouble, which since he is sixty years old is very probably of the hypertensive type.

His father's death from Bright's disease has some significance. We cannot always distinguish nephritis from hypertension without nephritis. Vascular disease of the kind that produces hypertension certainly runs in families. So even though this statement is about Bright's disease I think this family history has some bearing, which rarely turns out to be of any use.

We can see the marks of the questions asked as they get this history. They think a man with hypertension ought to have nosebleeds. They ask him about nosebleeds. They think he ought to have headaches. They ask him about headaches. And they get them.

"A left renal or ureteral stone." Considerable study on somebody's part is needed to make such a diagnosis, although it is of course a perfectly possible diagnosis.

A good many of us have had breathlessness after shoveling snow. I do not think that means much. "Palpitation following severe exertion" again does not mean much.

NOTES ON THE PHYSICAL EXAMINATION

What is the difference between brawny edema and soft edema? Brawny edema means worse and more of it. If we watch edema come, it is first soft, then the tissues get more and more stuffed with the fluid and so harder and harder. Brawny means extreme, then.

The apex impulse of the heart was not felt, presumably because of the shape of the chest. There is no evidence by percussion that the heart is enlarged. "Aortic second sound barely heard." That is very surprising, because the sort of disease we are thinking of produces a very sharp, clear aortic second.

The P wave is the important thing in this electrocardiogram. Certain variations in the P wave are very important as evidences of myocardial damage.

We have the clinical evidence of fluid in the chest as part of his general dropsy. We are not quite sure whether he had ascites or not, but probably he did.

The dullness below the right costal margin can probably be assumed to be due to the liver.

Hyperesthesia of the soles of the feet is not a symptom of anything that I know. Limited motion of the knees does not mean much at his age. Many healthy men have it.

The examination of the fundi is rather surprising. One would expect more change.

The gravity of the urine shows that he has no hyposthenuria, that inability to concentrate urine which goes so often with chronic nephritis. He can concentrate. There is no good evidence I should say, of kidney disease. We can easily have a renal function as low as this with perfectly functioning kidneys. We have had such a figure here in many cases which at post mortem were found to have normal kidneys. Many books have given the normal much too high, so that I emphasize this.

I cannot see anything important in this blood examination. Unless they measured the cells I cannot attach significance to that macrocytosis. Macrocytosis generally goes with pernicious anemia, rarely goes with anything else.

The non-protein nitrogen is a little high, but not high enough to be called abnormal.

The blood and urine, I should say, are essentially normal.

This is not a good x-ray plate, presumably because the patient could not hold his breath, had to move his chest, and obviously it is not a seven-foot plate, which is the only one we can be sure of as to the size of the heart. Nevertheless I shall be greatly surprised if we do not find that the heart is enlarged. From this plate I believe it is, although our percussion does not show it. If we cannot feel the apex impulse we are never sure of the cardiac size unless we have the x-ray. Palpation and x-ray are the only reliable methods we have, percussion, as we have often seen, being quite unreliable.

No interpretation is given of the report on the lung markings, but my guess is that they said what we should say, passive congestion of the lungs from hypertrophy of the heart. Prominent lung markings come from congestion of the lungs, more blood in that part and less air; all the rest of the report is natural in such a case as this, i. e., is due to congestion.

I have long ago given up trying to identify murmurs over the tricuspid area. I do not believe any human being can do it.

He lost twenty pounds of edema, of course. "He suddenly became irrational." That sequence of events is quite common. Loss of edema in one way or another, by tapping, by purgation, by diuretics, is quite often followed by mental symptoms. We do not know the reason. We conjecture that whatever toxic products were previously in the system became concentrated by the taking out of all this edema which before that kept them diluted and so more or less harmless. One always has to bear in mind that possibility, the following of mental symptoms on the quick reduction of edema.

I can not make anything of those signs on February 7. Egophony is one of the most unreliable and useless of signs. If we know perfectly well what we have we can sometimes verify it by egophony, but if we have no idea what is the matter egophony only throws dust in our eyes. I have seen it used to distinguish between fluid and solid in the lungs, and it generally fails. I mean by egophony a particular nasal type of voice heard in a particular place in the chest, when the places around it do not give that tone. The resemblance to the voice of a goat is not striking.

The patient was in the hospital only about two weeks, the average stay, and did very well or he

would not have been allowed to go out so soon. He must have been warned against stair climbing. He must have done it contrary to advice. I cannot imagine his leaving this hospital without being warned against that. He is now getting towards orthopnea. Presumably he had been given digitalis in the hospital and had kept on taking it in small quantities.

Cheyne-Stokes is a new sign, or at least was not mentioned before. They should have looked for it and presumably did. It is important in relation to the prognosis, although it has no particular significance in diagnosis. We always fear death within a few weeks or months when we get a Cheyne-Stokes respiration.

It is ever so much easier to put down these percussion measurements after we know by x-ray that the heart is good and large.

I take it there was no alternation. When we are looking for that very bad prognostic sign, the best way is in taking the blood pressure to see whether all the sounds heard through the stethoscope have the same volume, or whether every other one is louder. I think that is what they are looking for here when they say "the sounds were of uniform quality",—that is, the sound heard with the stethoscope over the artery.

This blood examination gives the first sign of leukocytosis we have had, because the polynuclears were low before.

Did the temperature go steadily up?

Miss Painter: It was 101° at entrance, went down to 99°, and then up.

Dr. Cabot: It was up most of the time then, we will judge. Of course we are thinking of some terminal infection here, and the temperature is important in relation to that.

DIFFERENTIAL DIAGNOSIS

Although we have much detail about this case, it all points in one direction. It seems like a simple case. If it is not it is going to be our fault. The underlying condition, surely we must say, is heart disease. What kind of heart disease? Think of the four types of heart disease,—rheumatic, syphilitic, hypertensive, coronary. There is no evidence of coronary or of rheumatic, or of syphilitic except one Wassermann,—no good evidence. Hypertension is certain because he has that high blood pressure and because there is nothing else to cause it. Merely the finding of high blood pressure does not prove heart disease. We can have it from brain disease. But we have no evidence of brain disease. So hypertension plus the absence of evidence of any other cause for it means hypertensive heart disease. That means that the heart ought to be hypertrophied and dilated. There should be a certain amount of arteriosclerosis, passive congestion of his lungs and liver, hydrothorax and ascites.

All of that we need not discuss. The question is of terminal infection and of the possible connection of that with the musical systolic murmur which was heard the first time he was here, and with the questionable systolic thrill felt the second time he was here. Both of those things suggest acute endocarditis, acute vegetations. But they do not prove it in any way, and it is very difficult to make that diagnosis unless one has help from embolism and blood culture. We have nothing to suggest either embolism or an infarction of the blood stream, so we may guess at the existence of an acute vegetative endocarditis, but we cannot go further. As to other terminal infections, there is nothing to give us a clue. The commonest is pneumonia, and pneumonia without any signs would be the first thing to think of. The second commonest is pericarditis. Pericarditis often comes without any signs. It is perfectly possible in a case like this to find terminal plastic pericarditis. Acute endocarditis is the third thing I should think of, and beyond that it is hardly possible to make a guess.

So he ought to have hypertensive heart disease with all that goes with that in the way of hypertrophy and dilatation and passive congestion. He has probably, not certainly, some terminal infection, pneumonia the first, pericarditis the second, and acute endocarditis the third possibility.

A Student: Could you say that death was caused by pulmonary edema in this case?

Dr. Cabot: No, I do not think I could. He has fluid in his chest, but there are many other factors in the case, so that one cannot put that as the cause of death. I should say his death was probably due to a combination of passive congestion (in the lungs and elsewhere) and infection, which often weakens the heart.

A Student: What are the possibilities of myocarditis?

Dr. Cabot: They are perfectly good. But we cannot make a diagnosis except on the basis of electrocardiographic evidence, which is suggestive but not conclusive here, or on evidence of cardiac infarction which ordinarily comes with fibrous myocarditis.

A Student: Don't you think that with arteriosclerosis and hypertension there would be apt to be some kidney involvement?

Dr. Cabot: I do not think there is going to be any nephritis. That line between renal arteriosclerosis and nephritis is one that pathologists do not all draw in the same place, and the same pathologist does not draw it in the same place all the time. I think this case will show arteriosclerosis in the kidneys as it will elsewhere, but not the particular changes in the smaller arteries that should go with vascular nephritis.

A Student: What other signs would you require for nephritis?

Dr. Cabot: In the first place fixed gravity of the urine, in the second place high retention products in the blood. We have not either of those, and those are the two most important things that I know.

A Student: A certain physiologist says that a person never dies of disease of the heart valves, but of myocardial failure. It is certainly true that myocardial failure means that the heart stops. How do clinicians look on that?

Dr. Cabot: I think clinicians think it is just a quibble over term. In some of the nursery rhymes one hears of dying from want of breath, and so one hears of myocardial failure in the sense of the heart's stopping. But in many cases of heart disease the myocardium is in splendid condition after death, macroscopically and microscopically, so we have no evidence of death from myocarditis. If one says he died of a tired heart, yes; many people die of tired hearts. But I do not like to put that into a list of diseases.

A Student: Is not the hypertrophied and dilated heart of hypertension called myocarditis?

Dr. Cabot: It is, but it should not be so called. Hypertension usually goes with a big, beefy heart, but often without any myocarditis. Nevertheless, it has been called myocardiits by some teachers of this school.

INTERPRETATION OF X-RAY

The findings suggest generalized enlargement of the heart, arteriosclerosis and chronic passive congestion.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Hypertensive and arteriosclerotic heart disease with decompensation.

DR. RICHARD C. CABOT'S DIAGNOSIS

Primary hypertension.
Hypertensive heart disease.
Hypertrophy and dilatation of the heart.
Arteriosclerosis.
Arteriosclerosis of the kidneys.
Chronic passive congestion of the lungs and liver.
Hydrothorax.
Ascites.
Terminal infection—(1 pneumonia, (2) pericarditis or (3) vegetative endocarditis.

DR. JOHN BRADLEY

ANATOMIC DIAGNOSIS

1. Primary disease.
   Arteriosclerosis (arteriolar) with arteriosclerotic nephritis.
2. Secondary or terminal lesions.
   Hypertrophy of the heart.
   Fibrinous pericarditis (slight).
   Bronchopneumonia.
   Central necrosis of the liver.
   Chronic passive congestion.

The pathological findings serve to confirm the clinical deductions.

The immediate cause of death was the heart. It weighed 590 grams. Both ventricles were greatly hypertrophied and considerably dilated. There were atheromatous plaques on the mitral valve, but no suggestions of a rheumatic lesion, a slight degree of calcification in the aortic cusps, but nothing was found to warrant a diagnosis of any valvular lesion. The tricuspid valve was negative. The coronary arteries were capacious and the intima normal except for rare atheromatous patches. The aorta showed a relatively slight degree of sclerosis.

The other organs showed the usual changes resulting from cardiac insufficiency. The peritoneal cavity contained nearly a liter of clear straw-colored fluid, the right pleural cavity being obliterated by adhesions. The liver showed chronic passive congestion and central necrosis.

In the kidney, however, the capsules stripped with slight difficulty, leaving a finely granular surface. The tissue was of unusual firmness, and on microscopical examination the smaller blood vessels showed intimal thickening and hyalin changes in the media. There were many groups of sclerosed glomeruli with tubular atrophy. The pathologist in this case made a diagnosis of arteriosclerotic nephritis with general arteriosclerosis, hypertrophy of the heart, central necrosis of the liver, and chronic passive congestion.

Dr. Cabot's deductions as to terminal infection are well borne out. The lungs, in addition to deep congestion and edema, showed an early bronchopneumonia, and the pericardium a slight fibrinous pericarditis.

Dr. Cabot: Of course I do not for a moment doubt this to be a definite nephritis and my own diagnosis to be wrong. What I will ask you to watch for during the year is this: we shall see other cases with exactly such a urine as that, with all the other facts the same, and the kidney called normal post mortem. In other words, I do not think we are in possession at this time of any clinical type of nephritis. Sometimes we recognize it, but it often goes with cases like this when there is nothing wrong with the kidney clinically.

A Student: Why is the functional test of so little value?

Dr. Cabot: Because of so many other factors besides the kidney in it, the difficulty of circulation for instance. This is a test where we inject something subcutaneously. A block in the kidney is only one place in which it can be retained. The metabolism, the rate of infiltration through the tissues, makes all the difference. For a good while I used to think that fifteen per cent was rather dangerous until I had seen so many cases with a function as low as that or even lower with the kidney found normal. Of course I do not think we ever get anything like that in health. It is always in sick people, but not always in nephritis.

As long as the question ofmy ocarditis was raised I have asked Dr. Bradley about it. He says there was no myocarditis.

TRUTH ABOUT MEDICINES

NEW AND NON-OFFICIAL REMEDIES

Solution of Invert Sugar-Lilly. — A solution of a misture of dextrose and levulose, obtained by the inversion of sucrose. Solution of invert sugar-Lilly is used in the injection treatment of varicose veins. It is claimed that the use of sugar solutions such as solutions of dextrose or of invert sugar have the advantage over solutions of sodium chloride, sodium salicylate or mercuric chloride in that they do not cause severe cramps or sloughing if accidentally injected outside the veins. Solution of invert sugar-Lilly is marketed in ampules containing 5 Gm., 6 Gm., and 7.3 Gm., respectively, in 10 cc. Eli Lilly & Co., Indianapolis.

Propaganda For Reform

Toxogon.—Toxogon is the therapeutically suggestive name applied by the Von Winkler Laboratories, Inc., Chicago, to a preparation proposed for the treatment of infectious diseases, particularly gonorrhea. The Council on Pharmacy and Chemistry found Toxogon unacceptable for New and Nonofficial Remedies because its composition was not adequately declared; because no evidence was available to indicate that the composition and uniformity of the product was controlled; because the claims advanced for it were unwarranted in the light of the available evidence; and because it is marketed under a therapeutically suggestive name. When the Council's report was submitted to the Von Winkler Laboratories, a reply was received which submutted further information but which did not permit a revision of the rejection of Toxogon. (Jour. A.M.A., November 2, 1929, p. 1383).

Undulant Fever.—A specific treatment of undulant fever is not yet available. The use of serums has proved disappointing. Vaccines have given more encouraging results according to recent reports from the continent. In particular, an antigen prepared from dried **Brucella abortus** has seemed efficacious in a small number of cases. In this country the use of acriflavine hydrochloride has been suggested to shorten the duration of the disease. (Jour. A.M.A, November 9, 1929, p. 1475).

Potency of Arsphenamine.—There is no official standard for therapeutic potency of arsphenamine preparations. According to reports of the United States Public Health Service Hygienic Laboratory, no one brand has been definitely established as superior to others when considered from the point of view of clinical efficiency. In some foreign countries, every preparation of arsphenamine and neoarsphenamine is tested on mice for therapeutic efficiency before being used. (Jour. A.M.A., November 9, 1929, p. 1495).

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BEGINNING VOLUME FOURTEEN

We feel that the accomplishments to be recorded in Volume XIV of SOUTHWESTERN MEDICINE will be notable in the medical annals of the southwest. We have every reason to expectthis and wish to recall some of these reasons to the attention of our readers.

Our ability to publish a first class journal for the profession of the southwest is largely due to our affiliation with the Cooperative Medical Advertising Bureau,—a subsidiary of the American Medical Association. Affiliation with the Bureau means that no products are advertised in this journal which are not approved by the Council on Pharmacy and Chemistry; therefore, our readers can safely depend on any product which appears in our advertising pages. Institutions and commercial houses which advertise in these pages must also be operated according to the high ethical standards required by the best ideals of the medical profession. Most of the revenue which makes this journal possible comes from the advertisers secured for us by the Bureau. Loyal supporters of this journal will remember this.

During the past five years the journal has been published by the A. C. Taylor Printing Company, and the association has been very pleasant. Mr. Taylor takes a personal interest in advancing the journal, acting as Advertising Manager. During the past year, we exceeded our allowance of reading space by about fifteen pages, for which the publishers made no extra charge. They will continue to punblish the journal this year.

Our editorial staff has been very loyal, and their efficiency can be measured by the amount of material from each section which appears in the journal. Dr. Orville H. Brown of Phoenix represents Arizona; Dr. Leslie M. Smith of El Paso has represented western Texas; and Dr. L. B. Cohenour of Albuquerque has represenetd New Mexico.

There is an abundance of material coming to the journal, and constantly we are forced to reject papers offered us, because to our minds, they do not come within the established purpose of this journal. That purpose is to reflect the medical accomplishments and medical thought of our own constituency, or to publish those papers read before our constituent organizations. During the coming year, we expect to continue this policy, and hope that the accomplishments will be even more worth recording than they were during the past year.

EL PASO COUNTY MEDICAL SOCIETY

The El Paso County Medical Society held their annual banquet on December 9th, at which time the following officers were elected for the year 1930:

President,—Dr. Paul Gallagher.
Vice-President,—Dr. J. W. Laws.
Secretary-Treasurer,—Dr. S. H. Newman.
Associate Editor for Southwestern Medicine,—Dr. James J. Gorman.
Librarian,—Dr. F. C. Goodwin.
Censors,—Drs. G. Werley, W. W. Waite and S. D. Swope.
Delegates to Texas State Medical Association,—Drs. W. L. Brown and T. J. McCamant; alternates, Drs. W. W. Waite and R. L. Ramey.
Delegate to American Association for the Advancement of Science,—Dr. E. C. Prentiss.

GOOD SAMARITAN HOSPITAL (Phoenix) OCTOBER STAFF MEETING

The Medical and Surgical Staff meeting of the Good Samaritan Hospital met Monday evening, October 28, with 32 present.

The chairman of the staff, Dr. Thayer, presided. He announced that no October meeting of the Council had been held and therefore the first order of business was election of officers.

The nomination and election were both by ballot. The two receiving the highest number of votes were declared the nominees, unless one had a majority on the first ballot.

For chairman of the staff, Dr. H. P. Mills and Dr. L. H. Thayer were the nominees. The chairman, Dr. Thayer, asked that a motion be made from the floor declaring Dr. Mills unanimously elected chairman of the staff. Such a motion was made by Dr. McIntyre. It was duly seconded and carried. Dr. Mills was declared president of the staff for the ensuing year.

Dr. Brown announced that he did not wish to be a candidate for the office of secretary as his duties made it impossible for him to do justice to the office. The ballots were then cast for the office of secretary. The two niminees were Dr. S. I. Bloomhardt and Dr.O.H. Brown. Dr. Brown asked that his name be withdrawn and that a motion be made to declare Dr. Bloomhardt unanimously elected secretary. Dr. Jordan made such a motion. It was duly carried and Dr. Bloomhardt was declared elected secretary for the ensuing year.

The scientific program was then taken up. Dr. Bloomhardt, chairman of the records committee reported upon the deaths for the past month as follows:

CASE 6863

Cerebral hemorrhage, female, age 55. One week before admission she had general weakness, a listless condition, generalized pain, considerable nausea and vomiting of bile, with nervous twitching of her muscles; she grew progressively worse. She had had kidney trouble of many years standing. Eighteen years ago a diseased kidney was removed; she also had an intestinal operation. She lived fairly comfortable following these operations with adherence to strict diet. She was brought to the hospital in practically a moribund state, with auricular fibrillation, secondary to myocardial insufficiency. The spinal fluid cell count was 75, no T. B. nor other organisms. The urine had positive albumin, many pus cells but no casts. W.B.C. 12,500, 90 per cent polys. The blood pressure was not charted. This is a fairly good history and physical. Blood pressure should have been charted.

CASE 6696

Male, age not noted, with uremic coma and aneurism. Unable to obtain history as patient was in coma; consultation, but no record was made of it. The non-protein nitrogen was 50 mgm. The urine had a trace of albumin, 6-10 blood cells and many pus cells.

CASE 6693

Male, age not recorded, diagnosis of pulmonary tuberculosis. He had severe pulmonary hemorrhage two days before admission. Two brothers had died of pulmonary tuberculosis. His disease was of six years standing. He died the day of admission. Excellent history and physical.

CASE 6707

Female, age 85, with chronic nephritis and coma.

The surgical treatment of chronic nephritis by decapsulation of the kidneys is a subject which is discussed recently and is interesting. It is too new to pass an opinion upon it. In a recent report from Guys Hospital, Dr. John Fawcett states that the group of cases most favorable in its employment is that in which, despite previous treatment, there is persistent edema accompanied by a large quantity of albumin and cases, commonly called parenchymatous nephritis. Even in this group, relief is all that can be expected. The terminal results, however, have often been found to be of real and lasting value to the extent of allowing the patients to resume work. Despite its disadvantages, Fawcett maintains the opinion that we are fully justified in resorting to this operation within certain limits. In advanced cases of chronic interstitial nephritis decapsulation should not be performed, for it is not only useless, but dangerous. While Fawcett is in accord with this statement, he qualifies it to the extent of saying that any evidence of interstitial, renal, cardiac or vascular disease must not be considered as an absolute contraindication to the operation. In one of his cases, such interstitial changes were beginning, but the patient did well after the operation.

CASE 6642

Male, age 53 with myocarditis and chronic nephritis. Four weeks before admission, arms, legs and feet began to swell; there was no history of other illness.

Autopsy revealed anasarca, two quarts of fluid in the abdominal cavity, free fluid in pleural cavities, atrophy of liver, infarct of right lower lung, heart hypertrophied and diffuse parenchymatous nephritis.

CASE 6737

Male, age 50, with appendicitis and streptococcic septicemia. He gave history of generalized pain over abdomen. He has been troubled for considerable time with indigestion and constipation, for which, during the past three months, he has been living on oranges, eating as high as two dozen per day. Very brief physical.

Operation the day after admission revealed abscessed retrocecal appendix. This was removed. Abscess was in meso-appendix; this was opened and drained. Temperature receded gradually from 103 to 99. It then started to run septic on the fourth day; this was accompanied by chills on the 4th, 5th, 6th postoperative days. Leucocytes mounted daily with increased percentage of polys. There was no tenderness in abdomen pointing to localization. The blood culture was negative. Widal and malta tests and x-ray of the chest were all negative. A Los Angeles physician was in consultation and he thought there was a deep abscess either in appendiceal region or in the region of the liver. A second operation failed to disclose any localized abscess. Following this, temperature ran to 106. Blood culture was again negative. Following day temperature was 107 and death resulted. An extremely inetresting and well-worked up case—one of those heart-rending cases which cause gray hairs.

The seriousness of appendicitis as a professional problem is indicated by recently collected figures from the Bureau of Vital Statisitcs which show that 25,000 persons die annually in the United States from the acute form. These figures, even if only approximately correct, suggest that no member of the profession has a right to rest on the laurels of his record, but should be urged to a more intensive study of the whole problem of acute appendicitis.

In regard to the type of case just reviewed, a large amount of study is being done and these studies lean toward the thought that inflammations of the appendix are consistently paralleled by changes in the course and character of the blood vessels and it is believed that mesenteriolitis, beginning as a collateral involvement, may later constitute an infection independently of the original appendicitis. Feriz studied 953 cases from anatomical, pathological and bacteriological basis with view of involvement of appendicular mesenteriolitis and noted the following phases: Hyperemia, edema and early damage of the blood vessel wall; at a more advanced state, lymphangitis and lymphadenitis—diffuse and local serocellular infiltration and pus formation; the infection then mostly subsides but may

give rise to: (1) Injection of the mesenteric vessels, interfering with the blood supply of the appendix and leading to gangrene; (2) an ascending septic thrombosis leading to pyelophlebitis and multiple hepatic abscesses; (3) diffuse peritonitis. He found microorganisms in 47 per cent of his acute cases with an average duration of eighteen hours. Streptococci appeared to play the main role. Complications due to mesenteriolitis may arise even after the appendix has been removed. Supporative mesenteriolitis is clinically manifested by a chill or multiple chills. Some of the best men are advocating a ligation or better resection of the ileocolic vein as a routine measure in this type of case. If the diagnosis is made before the operation, the ligation is done before the appendix is removed. Personally, I should think, in a matted up mass which is likely to be encountered in these cases, the procedure would be difficult and endanger the patient, but it is being done and with a lessening of mortality.

While on the subject of appendicitis; that of the chronic form is being discussed more and more in the literature. In short, at present, it is not a popular diagnosis among our foremost surgeons. Carnett gives the opinion: Chronic appendicitis will soon be generally non-existent as it is now claimed to be by many of our best pathologists, or as an almost universal affection due rarely to a preceding attack of acute appendicitis but most commonly the result of degenerative changes which are incident to increasing age and develop too gradually to cause clinical symptoms. Lichty even advises against making a diagnosis of chronic appendicitis in the absence of a definite history of characteristic acute attacks. He believes that poor results from operation for chronic appendicitis represent not operative failures but diagnostic mistakes. Stanton divides his cases of failure into two groups: (1) Those of young women complaining of right inguinal pain which is usually associated with constipation; and (2) those in which appendectomy was done unsuccessfully for the relief of vague abdominal symptoms.

The following cases were reported and discussed:

CASE 6772

DR. EDWARD HOEHN of Buckeye. Male, age 42. Farmer. Had been ill one week when first seen. He complained especially of shooting pain through the abdomen. A few days before he had been nauseated and vomited. The morning of the day on which he was first seen he had extremely severe pain in the region of the appendix. He stated that he had had pain in this area off and on for 20 years or more.

Usual childhood diseases; influenza three or four times during the past five years; poor health for many years and had been subject to attacks of dyspnea and nervousness.

Head and neck essentially negative. The heart was irregular. Blood pressure 120-110/80. Abdomen extremely obese with definite tenderness over McBurney's point; rigidity was not marked. Other findings were essentially negative.

He entered the hospital on Sept. 25 with diagnosis of acute appendicitis, and operation was done the same day. An ulcer on the intestine, almost to the point of rupture or ruptured slightly, presented itself. This was excised. An end to end anastomosis of the bowel was made; a cigarette drain and rubber tube were left in the abdomen.

The pathological report reads as follows: "Microscopic sections show the intestinal mucosa intact, except at one small point where the glandular structure appears desquamated. There is a slight inflammatory infiltration of the walls and a recent exudate on the peritoneal surface." Diagnosis was acute peritonitis with possible ulcer.

Following the operation the patient had persistent vomiting with relatively little tenderness in the abdomen. The urine was negative, except for an occasional pus cell. Following the operation the temperature gradually rose to nearly 104. The pulse rose rapidly from 88 to 140. The patient died on the fourth day following the operation. An autopsy was not obtained. It is believed that myocardial degeneration was largely responsible for his death.

The ultimate conclusions of the case were that he had a mild typhoid and that the ulcer was the result of the typhoid, and had the heart been normal the patient would probably have recovered.

CASE 6679

DR. H. J. FELCH. Male, 33 years old, came in complaining of general weakness and a constant feeling of tiredness. He stated that prior to 17 months ago he was a strong, vigorous man and could do any kind of heavy labor without fatigue or worry. He then moved to Durant, Okla. This was a swampy region where there were many mosquitoes. Following this he began to have chills and fever and was tired and weak. The chills were not regular in appearance. They came one or two a week. These have reoccurred for the past 17 months. He has taken medicine from various physicians at one time or another but has not been greatly benefited. He had never gone to bed before this for any trouble. His appetite was ordinarily good and his bowels regular. He has had the usual diseases of childhood. He had been in good health until 17 months ago. He was married; his wife died from child birth. He has two boys living and well. Other history is negative.

White male, about 40 years of age, with fair muscular development and nourishment; dull, sleepy appearance. The skin was dry and slightly bronzed. The conjunctivae, eyelids and lips are pale. The pupils react to light and to accomodation. Chest shows equal expansion on the two sides. Breath sounds are normal; there may be slight impairment of both resonance and sounds at bases. Heart is slightly enlarged, sounds distinct, no murmurs and no accentuations. The abdomen is moderately distended; organs not palpable and no masses; no pain and no tenderness. The G. U. system, glandular system and extremities are negative. The reflexes are slightly exaggerated. The urine showed an occasional pus cell but was otherwise negative. Red blood count 2,700,000, hbg.40, leucocytes 11,200, 48 polys, 52 monos; reds were crenated and marked poikilocytosis with malaria plasmodia present. The Wassermann was negative. The temperature was not high at any time, never going over 99.4. It however usually reached 99 on each day. The patient was given 10 grains of quinine three times a day. He made a gradual improvement and left the hospital on the sixteenth day.

DR. GOSS said that malaria was rarely seen in this valley but that it is occasionally found. It is often of the chronic type which makes it difficult to diagnose. The plasmodia are often not found in the blood even though the disease exists. He then introduced DR. C. W. BROWN, who said that he had had considerable experience in the treatment of malaria. This was in Arkansas, where he had lived for a number of years. He said that arsenic was the agent of choice in the chronic cases as the plasmodia have a tendency to get resistant to the effect of the quinine. The arsenic renders them more susceptible to quinine.

TESTING WEIGHTS
AND MEASURES

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DR. O. H. BROWN said that he had had a considerable experience in the treatment of malaria while on the visiting staff of the St. Louis City Hospital. He had received his early medical training in the University of Chicago and there he gained the impression that malaria was a disease which a layman could both diagnose and treat. The general belie was that malaria produced a chill every other day and that a few doses of quinine always cured the disease. This impression is far from the truth. Malaria is a most protean disease. He said that he had seen typical tertian malaria with chills and fever every third day as typical as any text book picture. Then he has seen tertian malaria simulate all other kinds of malaria and typhoid fever, tuberculosis, pleurisy and almost every other disease with which we are familiar. He has seen patients brought to the hospital after having had epileptic attacks and in coma and with bloody urine and many other conditions. All of these conditions may be due to malaria. He said he has seen them improved miraculously with a few doses of quinine. He has also seen them resist treatment for many weeks. Arsenic is of value in an occasional case but quinine long continued and in large doses ordinarily may be relied upon to cure malaria.

CASE 6744

Was reported by the secretary as DR. FAHLEN was not present. This was a woman 34 years of age, married. She came in with a skin eruption over her face, arms and exposed parts of the chest and back. There was intense itching. There was also swelling of the eyelids. For four days she had suffered with itching over the exposed parts of her body. She believes it is due to a nervous condition as she has recently been worrying over certain matters. She had had a previous attack a week before this, but it left in a short time. Four years ago she had a similar condition occurring with a nervous condition. Local treatment brought relief. She had the usual childhood diseases. She had influenza in 1917 and in 1925. She had an operation on her female organs in 1918. She began to menstruate at the age of 15. She menstruates regularly without pain, a moderate amount of three to four days duration. She has been married 19 years, and has three children. Her labors were all normal. She is troubled with constipation.

The physical findings were essentially negative except for the skin disturbance. There is an eruption by redness and edema of the skin and fine vesiculation with severe itching involving the face, neck, arms and dorsum of the hands. The involvement extends to the hair line of the scalp and over the exposed portion of the neck. The urine is negative except for a few pus cells. White count was 10,400,79 polys., and 21 lymphocytes. The temperature was normal. The patient was given a light diet, mainly fruit juices. Olive oil and cocoa butter were applied to the face for itching. She was also given castor oil.

The patient made a rapid recovery and was discharged from the hospital at the end of the sixth day.

DR. CLOHESSY said that the condition might be allergy or simply a speeding up of the normal reactions of the skin which finally caused violent reactions. He evolved a theory to the effect that any material applied to the skin frequently would cause a mild reaction; these reactions may become more pronounced with each application of the material. He hesitates to designate this as allergy. He recommends the use of one per cent solution of carbolic acid and zinc oxide ointment to the irritated places. He says anything stronger has a tendency to irritate the condition.

DR. O. H. BROWN said that it appeared to him as probably a case of ordinary food sensitization. He has had repeated experiences in which nervousness has produced a condition in which eczema and similar conditions result. The explanation which he offers is that the nervousness had stopped the digestive process and whole food or partially digested food goes from the alimentary tract to the blood stream and causes an effect upon the skin known as eczema. These cases are usually effectively treated by aids to the digestion and by changing the diet so that the person will not eat the foods which they have been accustomed to eat, and which are the probable causes of the eczema. He is

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Starch-free Diabetic Foods that are appetizing are easily made in the patient's home from Listers Flour. It is self-rising. Ask for nearest depot or order direct.
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For Excessive Armpit Perspiration
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The True Story of Acterol

CHEMISTS call it by its correct chemical name, *solution activated ergosterol*—the name by which we first supplied it.[1] The largest manufacturer of rare sterols in America, early having activated cholesterol[2] (1925), being first in America to commercially produce pure ergosterol[3] and to standardize activated ergosterol[1,4] (October, 1927), seeking to protect ourselves and the medical profession against substitution, we coined the name *Acterol*—signifying *activated ergosterol.* The Council on Pharmacy and Chemistry subsequently coined a name, *Viosterol.* As servants of the American Medical Profession, we defer to its wishes and now call our product MEAD'S VIOSTEROL IN OIL, 100 D. The product remains the same.

Therefore, so long as you specify

MEAD'S

call it Acterol, call it Activated Ergosterol
— call it VIOSTEROL IN OIL, 100 D —
so long as you specify Mead's,

You are sure of getting the original brand backed by the longest manufacturing and clinical experience. The paramount importance of this is evident from three striking truths: (1) We established the potency and (2) the dosage, both of which (potency and dosage) are now the official standards. (3) Mead's Viosterol does not turn rancid.

Specify Mead's Viosterol because it is accurately standardized, uniformly potent, free from rancidity, and safe to prescribe. Mead Johnson & Co., Evansville, Ind., enclose no dosage directions, and never exploit the medical profession.

[1] *J. Biol. Chem., 76:2.* [2] *Ibid., 66:451.*
[3] *Ibid., 80:15.* [4] *Ibid., 76:251.*

WATCH FOR SPECIAL COLOR SUPPLEMENT IN JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION JANUARY 18th, 1930

MEAD'S VIOSTEROL IN OIL, 100 D (*originally Acterol*). *Specific and preventive in cases of vitamin D deficiency. Licensed, Wisconsin Alumni Research Foundation. Accepted, Council on Pharmacy and Chemistry, A.M.A. All Mead Products are Council-Accepted.*

now using citric acid, 20 to 40 grains in a glass of water, taken with each meal and in the milder cases good results are immediate. In the severe cases it is frequently necessary to test the patient out for the foods which they are eating and to eliminate the worst reactors.

CASE 6609

In the absence of DR. COUCH, was presented by DR. KICKLIGHTER. This was a girl five years of age, brought to the hospital complaining of sore throat. She had had this sore throat for about 12 hours. Her appetite was poor. She had had a high fever for about 24 hours. She had always been in good health except for having had measles and mumps.

Examination revealed that the child was very restless at intervals, lying in bed with eyes crossed. She whines and calls for her mother. Seems to be in a mild stupor. Eyes react to light and to accommodation. Throat inflamed. Tonsils are greatly enlarged. The chest was normal. The heart sounds were fair but irregular. There seems to be a double beat about every fourth one. There was no distention of the abdomen and no palpable masses. The reflexes were exaggerated. The extremities were negative. A spinal puncture was done as soon as the patient entered the hospital and the spinal fluid revealed a cell count of 30,000 and meningococci were found on examination. The urine was negative except for a few pus cells and a very slight amount of albumin. The child was given anti-meningococcic serum and made a gradual recovery.

DR. MILLS, who assisted in the treatment of the case, said that in the early stage of meningitis intravenous administration of the serum is advisable but that in this case the daily administrations of the serum into the spinal canal had produced good results. He also believes that the serum we now have is more potent and more concentrated than it formerly was. He thinks there is rarely any good result from doing a cistern puncture. Occasionally there is a blocking of the canal so that a cistern puncture is necessary.

DR. MILLS reported experimental work which has been done showing that certain types of antimeningococcic serum works better in one case than in others.

The hour of ten o'clock having arrived the meeting was adjourned.

ORVILLE HARRY BROWN, Secy.

SITUATIONS WANTED

WANTED—Salaried appointments for Class A Physicians in all branches of the Medical Profession. Let us put you in touch with the best man for your opening. Our nation-wide connections enable us to give superior service. Aznoes National Physicians' Exchange, 30 North Michigan, Chicago. Established 1896. Member The Chicago Association of Commerce.

EL FRESCO REST HOME,
Los Angeles, California

Large comfortable house, with enclosed, furnished garden. Special emphasis on outdoor life and dietetics. Terms reasonable. References given. Address P. O. Box 1057, Station C., Los Angeles, Calif.

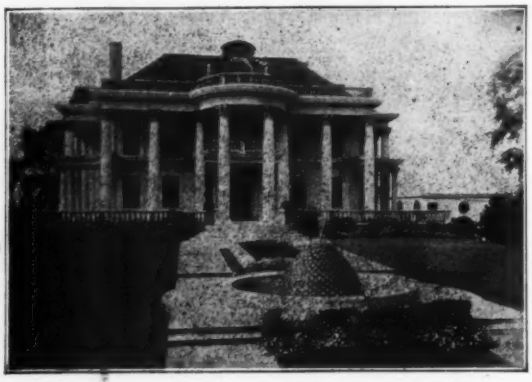
Founded 1896 by Dr. Hubert Work

WOODCROFT HOSPITAL, PUEBLO, COLORADO

New Building
New Equipment

NEURO-PSYCHIATRIC CLINIC
NERVOUS AND MENTAL DISEASES
DRUG ADDITIONS

H. A. LaMoure, M. D.
Superintendent



The G. Wilse Robinson Sanitarium and Neuro-Psychopathic Hospital

For Nervous and Mental Disorders and Allied Conditions
Alcoholism and Drug Addiction

Pleasantly located, on a beautiful tract of 25 acres. Buildings are commodious and attractive. Rooms with private bath are available.

Approved diagnostic and therapeutic methods used.

Occupational therapy, recreation and entertainment.

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When Ultraviolet is indicated

THE unfortunate part of the widespread publicity that ultraviolet radiation has enjoyed is that it has unwittingly impressed many with the idea that this form of energy is a panacea for human ills.

Because of this situation many physicians have become lukewarm on the subject of ultraviolet therapy. But they fail to appreciate the fact that the public is quickly learning the folly of self-treatment for any *abnormal* condition. The physician is still the only recognized authority who can determine whether ultraviolet is indicated or contra-indicated in a given condition, and what constitutes correct dosage. For those reasons, the thinking man still turns to his physician for advice and treatment based on a knowledge of what medical science has established.

Are you equipped for ultraviolet *therapy?* May we tell you about the most powerful source known for artificially produced ultraviolet radiations, to the exclusion of infra-red? In other words, ultraviolet radiation for ultraviolet therapy.

Victor Quartz Lamps are designed for use by the medical profession exclusively. They are so powerful in ultraviolet output that *promiscuous* use of them would be dangerous. A given dosage is administered in a small fraction of the time required with other types of apparatus. Thus, not only is the physician's time and that of his patient conserved, but the opportunity of accomplishing desired clinical results is greatly enhanced.

There is a goodly number of models of the Victor Quartz Lamp. Send for our new complete catalog, which will help you in making a selection of the outfit best suited to your particular requirements.

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Manufacturers of the Coolidge Tube and complete line of X-Ray Apparatus — VICTOR — *Physical Therapy Apparatus, Electrocardiographs, and other Specialties*

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A GENERAL ELECTRIC ORGANIZATION

PERSONALS AND NEWS ITEMS

DR. OSCAR S. BROWN, of Winslow, member of the State Medical Examining Board, was in Phoenix the first week in January, attending the quarterly meeting of the Board. Dr. Brown has recently returned from an extensive tour through Central America, including Mexico, Panama, Colombia and Cuba.

DR. JOHN W. FLINN, of the Pamsetgaaf Sanatorium at Prescott, has opened offices in Phoenix. He will be located at Suite 505 in the Goodrich Building, for office consultation in tuberculosis.

DR. ALONZO A. BROWNING, of Phoenix, has moved from his former location in the Luhrs Building. to Suite 204, in the same building.

HOTEL DIEU, (El Paso) MEDICAL AND SURGICAL STAFF has elected the following officers for the year 1930:

Chairman,—Dr. R. L. Ramey.
Vice-Chairman,—Dr. J. A. Pickett.
Secretary.—Dr. Will Rogers.

THE CITY-COUNTY HOSPITAL (El Paso) MEDICAL AND SURGICAL STAFF has elected the following staff for the yar 1930:

Chairman,—Dr. W. R. Jamieson.
Vice-Chairman,—Dr. S. H. Newman.
Secretary,—Dr. Sherrod Mengel.

THE MARICOPA COUNTY MEDICAL SOCIETY (Phoenix), at their annual meeting held on December 16th, elected the following officers for the year 1930:

President,—Dr. Robt. T. Franklin, Glendale.
Vice-Presiden,—Dr. J. M. Meason, Chandler.
Secretary-Treasurer, Dr. Victor Randolph.
Censors,—Drs. R. J. Stroud, (Tempe), J. M. Greer (Phoenix) and E. W. Phillips (Phoenix).
Committee on County Hospital, Drs. J. M. Greer and Howell Randolph.

CAMP
Supporting Garments

Comfort and Support with New Inner Pad Belt

Where scientific abdominal uplift and support are desired, this new Camp Inner Pad Belt (Model No. 913) serves admirably. With the Patented Adjustment attached directly to the soft inner pad, the belt provides for correct upward and backward support. This Adjustment makes manipulation easy and a stronger pull possible. The outer elastic section controls extra adipose tissue. The Inner Pad Belt insures maximum comfort with proper support. Dealers stocking these items add a service which customers will appreciate . . . and, at the same time, increase profit possibilities. Sold by surgical houses and the better drug stores.

Write for our Physicians' Manual

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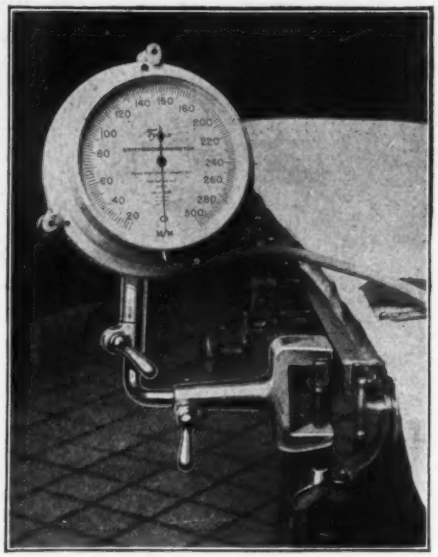
Manufacturers, JACKSON, MICHIGAN
CHICAGO 89 E. Madison St. LONDON 252 Regent St., W. NEW YORK 330 Fifth Ave.



Tycos Surgical Unit

For Blood Pressure Determination in the Operating Room

For the convenience of anaesthetists and surgeons, who are finding that accurate blood pressure readings are invaluable during anaesthesia and surgery, we have designed this *Tycos* Surgical Unit.

It consists of a large easy reading type *Tycos* Sphygmomanometer and a universal clamp. The clamp enables the Sphygmomanometer to be adjusted to any position convenient for the anaesthetist and out of the way of the surgeons and assistants. The adjustments can be made instantly, but once made the instrument is firm as the table itself. If it is inconvenient to have the instrument attached to the table, the clamp will accommodate it to the anaesthesia equipment or instrument stand.

Modern trends make it extremely important for hospitals to include the *Tycos* Surgical Unit in their operating room equipment.

Your dealer can supply you with this equipment. Complete unit $52.50. Clamp only $15.00. Write today for additional information.

Taylor Instrument Companies
ROCHESTER, N. Y., U. S. A.

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IN GREAT BRITAIN
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BOOK REVIEWS

PREVENTIVE MEDICINE:—By Mark F. Boyd, M.D., C.P.H., Member of Regular Field Staff, International Health Division of Rockefeller Foundation; formerly Professor of Bacteriology and Preventive Medicine in the Medical Department of the University of Texas. Third Edition, Revised. Octavo volume of 475 pages with 151 illustrations. Philadelphia and London; W. B. Saunders Company, 1928. Cloth, $4.50 net.

Dr. Boyd believes that the physician should play an important part in the field of preventive medicine and public health and that at present they are neglecting their opportunities. He also thinks that if the physicians continue to neglect this field it may be taken away from them by less competent, poorer trained, individuals.

It would seem that the field is of such importance that physicians may well afford to devote their time and energies to it. If the compensation is not adequate as yet, it is but a matter of having the public appreciate the full value of preventive medicine to get them to pay for it.

The vast amount of available material for this type of work has been greatly condensed. There has been attached to each chapter a list of references for supplementary reading.

The reviewer has no hesitancy in recommending this as one of the best works on the subject for the number of pages which it contains. O. H. B.

THE SURGICAL CLINICS OF NORTH AMERICA. (Issued serially, one number every other month). Volume 9, No. 1. (Mayo Clinic Number—February 1929) 247 pages with 141 illustrations. Per Clinic year (February 1929 to December 1929). Paper $12.00; Cloth, $16.00. Philadelphia and London. W. B. Saunders Company.

The fact that this is a Mayo Clinic number promises a most interesting volume.

An article which interested the reviewer more than others is entitled "The Postoperative Administration of Oxygen as a Prophylaxis Against Pulmonary Complications." In brief this article shows that the early administration of oxygen in pneumonia tends to lessen complications. If the oxygen can be administrated before the pneumonia develops it tends to prevent the development of the pneumonia. The oxygen has been administered mainly by means of oxygen rooms in which patient and nurse are kept for periods of several days. More recently they have administered it hypodermically and find that it seems to work in about the same way as when given through the lungs.

Other articles of interest are "The Significance of Urea" and The Phagocytic Cells of the Mammalian Liver, Pulmonary Tuberculosis, Empyema, and a large number of purely surgical subjects.

O. H. B.

THE TRUE STORY OF ACTEROL

To get the real facts on this important subject, do not fail to look for the special color supplement in the Journal of the American Medical Association for January 18.

In the meantime, please see the Mead Johnson announcement in this issue also entitled "The True Story of Acterol."

FOR SALE—Complete line of surgical instruments, operating table, two surgical tables. Was property of Dr. A. E. Lauson, deceased. Address Box 52, Anthony, New Mexico.

DOCTOR

The

Dallas Southern Clinical Society

Will Hold the Next Meeting

In Dallas, April 14, 15, 16, 17, and 18. There will be Lectures, Clinics and Post Graduate Work, covering Medicine, Surgery and the Specialties—this will be done in the forenoon.

Each afternoon will be devoted to General Meetings when men of national reputation will address you. Motion pictures will be shown, among them being the famous Canti (Cancer) film.

Remember the date, April 14-15-16-17-18

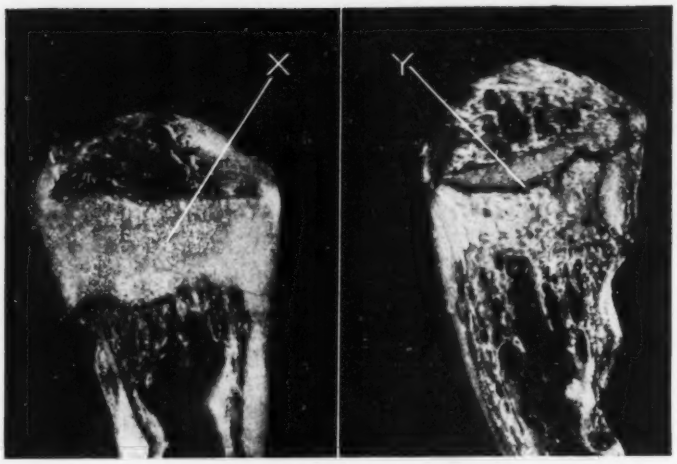
HEAQUARTERS, BAKER HOTEL

REGISTRATION $10.00 **DALLAS**

For further information write
Dallas Southern Clinical Society, 710 Medical Arts Bldg., Dallas

This Will be the Largest Clinic Ever Held in the South

BOTH Vitamins Definitely Measured



Illustrating "Line Test" method of standardizing Vitamin D content. At left, the leg bone of a rachitic rat showing induced decalcification area (X). At right, healing has begun, as evidenced by initiation of recalcification at dark line (Y).

How can vitamins be "measured?" What is meant by "standardized" when applied to Cod-liver Oil? Here, briefly, is the method followed in determining the vitamin content of Parke-Davis Standardized Cod-liver Oil:

To test for *vitamin A potency* the oil is given orally to young albino rats which have been fed on a diet free from vitamin A. We ascertain how much oil is needed daily to correct the induced typical eye condition (xerophthalmia) and to institute a specified rate of growth. The daily minimum amount of oil required to bring about this change constitutes one vitamin A unit.

Every lot of Parke-Davis Standardized Cod-liver Oil must contain not less than 13,500 units of vitamin A in each fluid ounce.

In determining *vitamin D potency* we use our quantitative adaptation of the "line test" technique of McCollum, Simmonds, Shipley, and Park. The oil is fed to young rats in which rickets has been induced. We measure the minimum amount of oil required per day over a period of ten days to initiate recalcification in the leg bones. This amount represents one vitamin D unit. Each fluid ounce of Parke-Davis Standardized Cod-liver Oil contains not less than 3000 vitamin D units.

Parke, Davis & Company was the first commercial laboratory to assay Cod-liver Oil for both vitamins A and D. Parke-Davis Standardized Cod-liver Oil is backed by years of research work in various phases of nutrition chemistry. Quite aside from its vitamin richness, this product has other distinguishing features which will appeal to you. It is clear, bland, and as nearly tasteless and odorless as a pure Cod-liver Oil can be. May we suggest that in prescribing Cod-liver Oil for your patients you specify the Parke-Davis product?

Send for stock package

To any physician who is personally unacquainted with Parke-Davis Standardized Cod-liver Oil we will gladly send a 4-ounce bottle for free trial.

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